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Integrating Emergency Care with Population Health

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Integrating Emergency Care with Population Health

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Emergency medicine is a specialty which closely reflects societal challenges and consequences of public policy decisions. The emergency department specifically deals with social injustice, health and economic disparities, violence, substance abuse, and disaster preparedness and response. This journal focuses on how emergency care affects the health of the community and population, and conversely, how these societal challenges affect the composition of the patient population who seek care in the emergency department. The development of better systems to provide emergency care, including technology solutions, is critical to enhancing population health.

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Does Targeted Education of Emergency Physicians Improve Their Comfort Level in Treating Psychiatric Patients?

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Introduction: We determined if targeted education of emergency physicians (EPs) regarding the treatment of mental illness will improve their comfort level in treating psychiatric patients boarding in the emergency department (ED) awaiting admission.

Methods: We performed a pilot study examining whether an educational intervention would change an EP's comfort level in treating psychiatric boarder patients (PBPs). We identified a set of psychiatric emergencies that typically require admission or treatment beyond the scope of practice of emergency medicine. Diagnoses included major depression, schizophrenia, schizoaffective disorder, bipolar affective disorder, general anxiety disorder, suicidal ideation, and criminal behavior. We designed equivalent surveys to be used before and after an educational intervention. Each survey consisted of 10 scenarios of typical psychiatric patients. EPs were asked to rate their comfort levels in treating the described patients on a visual analogue scale. We calculated summary scores for the non intervention survey group (NINT) and intervention survey group (INT) and compared them using Student's t-test.

Results: Seventy-nine percent (33/42) of eligible participants completed the pre-intervention survey (21 attendings, 12 residents) and comprised the NINT group. Fifty-five percent (23/42) completed the post-intervention survey (16 attendings, 7 residents) comprising the INT group. A comparison of summary scores between 'NINT' and 'INT' groups showed a highly significant improvement in comfort levels with treating the patients described in the scenarios ($P = 0.003$**).** Improvements were noted on separate analysis for faculty ($P = 0.039$) and for residents ($P = 0.012$). Results of a sensitivity analysis excluding one highly significant scenario showed decreased, but still important differences between the NINT and INT groups for all participants and for residents, but not for faculty (all: $P = 0.05$; faculty: $P = 0.25$; residents: $P = 0.03$).

Conclusion: This pilot study suggests that the comfort level of EPs, when asked to treat PBPs, may be improved with education. We believe our data support further study of this idea and of whether an improved comfort level will translate to a willingness to treat.
[West J Emerg Med. 2012;13(6):453-457]

INTRODUCTION

Crowding of emergency departments (EDs) and lack of acceptable available inpatient beds has resulted in increasing numbers of patients being held in the EDs after admission. Among this group are an increasing number of psychiatric boarder patients (PBPs) who await inpatient psychiatric treatment. These patients often languish in the EDs for days without receiving appropriate medication or behavioral therapy, while remaining in an environment not conducive to their recovery. The American College of Emergency Physicians, in collaboration with the American Psychiatric Association and the National Alliance for the Mentally Ill conducted an online survey to determine the number of psychiatric patients who were boarded in EDs in March 2004. Of 340 participating EDs, two thirds of the respondents reported increasing numbers of PBPs.[1] A National Health Policy Forum in August 2007 noted that crowding of EDs continues to be a significant problem and the ED lengths of stay of psychiatric patients were noted to be 42% longer than that for medical/surgical patients.[2] Slade et al[3] reported that the duration of EDs visit increased by 2.3% overall while that of mental health-related visits have increased 42%. A survey conducted at the University of Utah ED showed that psychiatric patients were more likely to be readmitted than medical patients within 30 days (21% versus 13.4%).[5] There was a 21.1% increase in state mental health admissions between 2002 and 2005 in 8 key states in the United States.[4] A recent study from California revealed a mental health system in crisis where increasing demand for inpatient psychiatric beds is being met with a diminishing supply. This has resulted in wait times for adult psychiatric patients exceeding 16 hours.[11] These phenomena may have been augmented by recidivism, emphasizing the urgency of finding a solution for this expanding problem.

The experience of our psychiatric faculty who frequently receive PBPs from outlying hospitals lead us to believe there is a significant subset of psychiatric patients who would benefit from earlier intervention. We believed that the increasing numbers of PBPs could be moderated by emergency physicians (EPs); however, we hypothesized that this was dependent on the "comfort level" of the EPs treating them. Comfort level is a vague quantity and one difficult to describe. For the purposes of this study we defined it as a feeling of ease and security on the part of the treating EPs when tasked with initiating care. It may already be within the scope of practice of emergency medicine (EM) to start appropriate medication for PBPs, who are boarding or will receive close follow up; however, the ability of an EP to act within this scope may depend on their level of comfort with these patients.

We performed a pilot study testing whether an educational intervention could improve an EP's comfort level in managing these patients. Our hypothesis was that focused instruction would improve the comfort level of EPs in caring for PBPs and thereby facilitate the care for this group of patients.

METHODS

The setting was a Southeastern, urban, academic medical center. We formed a study group consisting of faculty members from the departments of emergency medicine and psychiatry and a medical student with a career interest in emergency psychiatry. The psychiatric faculty member is dedicated to the treatment of PBPs and is considered an expert in their management at our institution. We designed 2 equivalents Data Collection Instruments (DCI) to be used before and after an educational intervention. Each DCI consisted of a survey describing the presentation of 10 emergency psychiatric patients designed to measure EPs' comfort levels in providing care to psychiatric patients in need of treatment that was beyond the ordinary scope of practice of EPs (see pre-intervention Survey: Appendix I, and post-intervention Survey: Appendix II). Scenarios described in the surveys referred to treatment of major depressive disorder, schizophrenia, schizoaffective disorder, bipolar affective disorder, generalized anxiety disorder, suicidal ideation, and criminal behavior (CB). Our scenarios were derived from our daily practice and represented the most common types of psychiatric emergencies one was likely to see in our busy academic practice. After administration of the pre-intervention survey we provided an educational intervention (Appendix III) designed to educate EPs about the nuances of treating the patients described in the scenarios. This consisted of a Microsoft PowerPoint 2010 presentation that explained the correct treatment of all patients described in the pre-intervention survey. The intervention was presented in a department conference and distributed to all eligible participants by email. After completion of the pre-intervention survey and the educational intervention, the post-intervention survey was distributed by email. Opportunity was provided for study participants to ask questions during the conference and via email to the investigators.

All practicing EPs at the study site were eligible to participate (42 EPs: 18 EM residents and 24 faculty). Because the participants who took the pre-intervention survey were not matched to those taking the post-intervention survey we decided that the most effective way to analyze our results would be to treat the pre-intervention and post-intervention groups as 2 cohorts: a non-intervention group (NINT) and an intervention (INT) group. Hence, though the samples comprising the 2 cohorts were drawn from the same population of EPs at our academic medical center, for the purposes of this pilot study we considered them as independent samples. Therefore, all comparisons were performed using Student's t-test. Our design was kept simple for practical reasons and we used convenience samples for both surveys. The participants did not receive an incentive to participate.

EPs were asked to rate their comfort levels in treating the patients described in the DCI on a visual analogue scale (VAS). We automated the surveys using a survey construction

tool (REDCap Survey Software - Version 1.3.5 - 2010 Vanderbilt University) and distributed them by email to all eligible participants with a letter assuring the voluntary and anonymous nature of the survey. Results were automatically collated by REDCap and uploaded into SAS, Cary, NC for analysis.

We calculated summary scores for the NINT and INT groups by adding the VAS scores for all individual scenarios in each survey. Summary scores for all participants were averaged to obtain an overall summary score for each group. We calculated separate summary scores for residents and faculty. The study was exempted by the Institutional Review Board of the Medical University of South Carolina.

RESULTS

Thirty-three of forty-two (79%) eligible participants completed the pre-intervention survey (21 attendings, 12 residents) and comprised the NINT group. Twenty-three of forty-two (55%) completed the post-intervention survey (16 attendings, 7 residents) and comprised the INT group. Comparison of overall summary scores between the NINT and INT groups showed a highly significant improvement in comfort levels (NINT mean 464.9, INT mean 580.7, P = 0.003)**.** There were also significant improvements in summary scores noted for faculty (NINT mean 500.9, INT mean 608.4, P = 0.039) and for residents (NINT mean 399.4, INT mean 517.3, P = 0.012) (Figure 1).

One scenario, the CB scenario, showed highly significant results for all categories of participants. We therefore performed a sensitivity analysis to determine if our results were driven by this 1 scenario. Results of the sensitivity analysis excluding the CB scenario showed decreased, but still important differences between the scenarios for the total group and for the residents; however, the data for faculty became non-significant (All: NINT mean 449.2, INT mean 520.5, P = 0.05; faculty: NINT mean 487.1, INT mean 544.1, P = 0.25; residents: NINT mean 379.3, INT mean 466.6, P = 0.03) (Figure 2). Tables showing the data for individual scenarios, summary score data, and data from the sensitivity analysis are provided in Appendix IV.

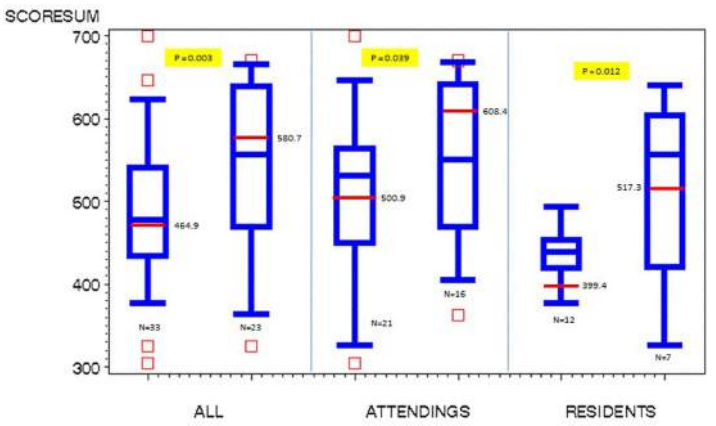

Figure 1. Non-Intervention (NINT) & intervention (INT) summary scores.

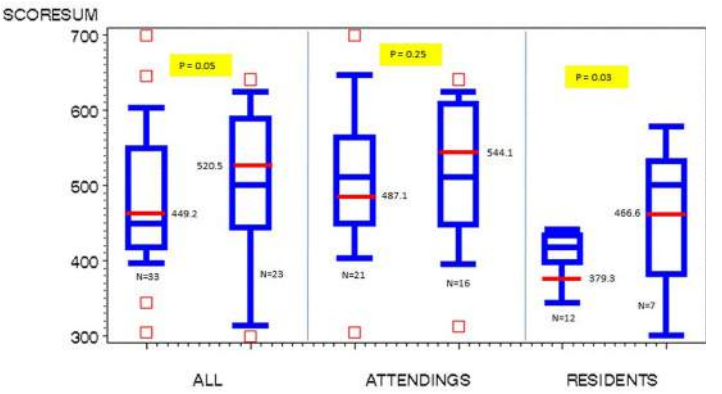

Figure 2. Non-intervention (NINT) & intervention (INT) summary scores with criminal behavior scenario removed.

DISCUSSION

The increasing demands placed on ED resources by PBPs suggest a need to review the current practice of EM. If PBPs received appropriate treatment while waiting for inpatient beds, many of them could improve sufficiently for discharge. Not only is this desirable for the patient, it is operationally crucial to EDs. National EM leaders have expressed serious concerns about the problem of boarder patients in the EDs, pointing out the compromised care and safety of these patients.[6] The demand for treatment of behavioral emergencies already exceeds the availability of services in many areas of the U.S. and with further cutback of mental health funding this problem will likely worsen.[7]

One solution we explored was the feasibility of training ED staff to manage PBPs. Previous studies have shown that ED staff and physicians can provide limited psychiatric care. Patel et al[8] demonstrated the effectiveness of a mental health risk-assessment tool that can be used in the EDs to organize the care of mentally ill patients more efficiently. Wulsin et al[9] found that EPs could successfully manage psychiatric patients and initiate therapy.

The financial impact of caring for PBPs is important for the patient and for the EDs. In many cases the ED is unable to bill for holding PBPs. One model to recover cost could be that of an ED psychiatric observation unit. Marchand et al[10] showed an ED Psychiatric Observation Unit to be cost-effective, resulting in reduced length of stay, without an increase in suicides and also facilitated rapid decision making and rapid referrals.

Our data suggest that it may be possible to improve the comfort level of EPs in caring for PBPs. The residents' comfort level showed a significant improvement in response to the educational intervention and, while faculty data did not show a statistically significant change, the direction of the data was in favor of a benefit from the educational intervention. This suggests that, regardless of its ability to change practice,

our presentation may be an effective teaching tool worthy of further study.

The improvement seen in the faculty data was driven by the results of the CB scenario. This scenario may represent an area where EPs have a particular need for additional instruction. The highly significant difference between the NINT and INT group scores for this scenario suggests a need for further training of EPs on how to manage patients with criminal behavior.

Our experience seems to show that one may successfully implement this educational intervention in an academic ED and that the potential exists for improving the comfort level of EPs in caring for PBPs. However, based on our findings we believe a future study of our idea with an improved design would be worthwhile. What is needed is a matched, controlled study that will allow us to differentiate the impact of our intervention from a placebo effect. Therefore, a future design should include pairing of participants between the NINT and INT groups. This would enable use of the paired t-test, providing a more effective comparison.

The survey instruments should be validated prior to the study using standard survey validation techniques. Despite their apparent equivalence, item pairs from the 2 surveys should be randomly assigned between the 2 data collection instruments to avoid introducing bias that may be inherent to the individual items. A larger sample obtained from multiple institutions would increase the generalizability of the data. Stratification of results by participants' years of training and practice would enable one to determine the effect of prior experience on our findings and help define subsets of EP who may experience particular benefit. Also, future studies should examine the relationship between EP's comfort levels and their willingness to expand their scope of practice to treat PBPs.

In theory, an EP willing to initiate care of psychiatric patients as they would for medical patients who are boarding in the ED could expedite care for these patients, improve ED flow, and reduce cost. We believe that further investigation of our idea using an improved design has the potential to benefit PBPs and streamline ED operations.

LIMITATIONS

Limitations of this study include the small sample size and its confinement to a single academic medical center. Not having validated the surveys using established survey validation techniques limits their ability to measure that which they claim. A lack of randomization in the order of administration of the survey items to participants in the NINT and INT groups introduced bias inherent to idiosyncrasies of the scenarios. Not matching the participants from the NINT and INT groups resulted in a lack of control for variation among participants regarding their practice styles, personalities, knowledge, and experience. Matching participants would permit us to differentiate the effect of the intervention from a placebo effect and improve accuracy by using a paired t-test. A failure to stratify responses by years of training and practice limits our ability to note the impact of these factors on the effectiveness of our intervention. Furthermore, "comfort level" must be distinguished from "willingness" when considered in terms of asking EPs to change their scope of practice.

We were concerned that one scenario, the CB scenario, which showed extremely high significance, might have skewed the overall data. We therefore performed a sensitivity analysis to address this problem. This showed that although one group, the faculty, was strongly influenced by this scenario, the overall findings still suggested that the intervention had merit. A larger sample and a better response rate may have produced more significant results. In future studies an incentive to participate may improve response rate.

Although our study instruments were not validated prior to their use in this study, our scenarios were developed through collaboration between an expert in emergency psychiatry who has practiced EM and is board certified in psychiatry and members of the EM faculty. Further studies are needed to corroborate our findings in a larger more varied setting.

CONCLUSION

This pilot study suggests that it may be possible to improve the comfort level of EPs when asked to treat psychiatric patients, such as those described in our scenarios, by using a focused educational tool. Whether our findings will stand up to more rigorous study and whether a change in comfort level will translate to an expansion of EPs' scope of practice remains to be seen. We believe that further study of this idea using an improved design is justified given the expanding population of PBPs.

Our findings also support further study of the effectiveness of our educational intervention as a teaching tool, particularly with regard to the CB scenario. The management of patients with criminal behavior appears to be an area where EPs would benefit from additional graduate medical education.

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Factors Affecting Candidate Placement on an Emergency Medicine Residency Program's Rank Order List

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Introduction: Several factors influence the final placement of a medical student candidate on an emergency medicine (EM) residency program's rank order list, including EM grade, standardized letter of recommendation, medical school class rank, and US Medical License Examination (USMLE) scores. We sought to determine the correlation of these parameters with a candidate's final rank on a residency program's rank order list.

Methods: We used a retrospective cohort design to examine 129 candidate packets from an EM residency program. Class ranks were assessed according to the instructions provided by the students' medical schools. EM grades were scored from 1 (honors) to 5 (fail). Global assessments noted on the standardized letter of recommendation (SLOR) were scored from 1 (outstanding) to 4 (good). USMLE scores were reported as the candidate's 3-digit scores. Spearman's rank correlation coefficient was used to analyze data.

Results: Electronic Residency Application Service packets for 127/129 (98.4%) candidates were examined. The following parameters correlated positively with a candidate's final placement on the rank order list: EM grade, $\rho = 0.379$, $P < 0.001$; global assessment, $\rho = 0.332$, $P < 0.001$; and class rank, $\rho = 0.234$, $P = 0.035$. We found a negative correlation between final placement on the rank order list with both USMLE step 1 scores, $\rho = -0.253$, $P = 0.006$; and USMLE step 2 scores, $\rho = -0.348$, $P = 0.004$.

Conclusion: Higher scores on EM rotations, medical school class ranks, and SLOR global assessments correlated with higher placements on a rank order list, whereas candidates with higher USMLE scores had lower placements on a rank order list. However, none of the parameters examined correlated strongly with ultimate position of a candidate on the rank list, which underscores that other factors may influence a candidate's final ranking. [West J Emerg Med. 2012;13(6):458–462.]

INTRODUCTION

There are several key components of Electronic Residency Application Service (ERAS) packets for medical students seeking to match to an emergency medicine slot, including preclinical and clinical medical school grades, emergency medicine (EM) clerkship elective grades, standardized letters of recommendation (SLOR), and scores on step 1 and step 2 of the US Medical License Examination (USMLE).[1] Another key portion of the application is the Medical Student Performance Examination (MSPE) or Dean's Letter, which is compiled using these data.[1] The Association of American Medical Colleges advises that the MSPE contain "a summative assessment of the

student's comparative performance in medical school relative to his/her peers, including any information about school-specific categories used in differentiating among levels of student performance."[2] The SLOR is a particularly important document for candidates hoping to match in EM. It is a letter of recommendation that is universally used by EM programs for medical students who request one. In addition to the student's clerkship grade, proficiency ratings for certain attributes, and comments from faculty members regarding the student's performance, the SLOR also contains a global assessment score (outstanding, excellent, very good, or good).[3,4]

Most medical student candidates for EM residency attempt to obtain high marks in all these categories, but does academic excellence as manifested by outstanding grades, competitive class rank (CR), high USMLE scores, or the highest global assessment on the SLOR really result in a top ranking on an EM residency program's rank order list (ROL)? Several articles outline the EM application process and the value of each part of the application packet.[1,5,6] Further, a survey of EM program directors published in 1999 revealed that EM rotation grade, interview, clinical grades, and recommendations were most important in the selection of residents and that program directors placed moderate emphasis on USMLE steps 1 and 2.[7] A more recent study which looked at the importance of criteria used for residency selection across 21 specialties found grades in required clerkships and USMLE step 1 scores were the most important factors for residency program directors.[8] However, we were unable to identify studies that evaluated the actual value of the various pieces of the residency application packet.

We sought to determine the correlation between EM grades, SLOR global assessment rating, CR, and USMLE steps 1 and 2 scores with a candidate's final placement on an EM residency program's final submitted National Residency Match Program (NRMP) rank list. Because there are no validated scoring instruments to assess the interview, we did not examine whether there was a correlation between an interview rating and final rank position.

METHODS

Study Design

This was a retrospective cohort analysis of the applications of residency candidates interviewed during 2007–2008 for matriculation into a single EM residency program for the 2008–2009 academic year. This study was conducted after interviews had finished and after the final NRMP ROL had been submitted. This study was reviewed by our institutional review board and deemed exempt from informed consent.

Study Setting and Population

This study was conducted at a single, 3-year EM residency program that sees approximately 160,000 adult and pediatric patients per year. All candidates for that program's 12 categorical EM positions were included in the analysis.

Study Protocol

All information contained in the candidates' ERAS applications at the time of their interview was examined after interviews for that year had been concluded and the ROL had been submitted. Application information was available for 127/129 of the candidates and was subjected to analysis. These candidate application packets were examined by a single data abstractor and were extracted onto a Microsoft Excel Spreadsheet (Redmond, Washington). The abstractor, who was a study author, was blinded to the results of the final rank order list.

A priori definitions were as follows: EM grade was noted from the SLOR or from the candidate's transcript. All listed EM grades were included. If more than one EM grade was noted, then all EM grades were used to calculate a composite score. Grades were noted in descending order with the top grade as 1 and the lowest 5. Grades marked in between were adjusted accordingly (eg, a candidate assigned an honors/high pass was given a 1.5). A final composite score was given to each candidate that was then used for correlation purposes.

A candidate's CR was determined by the MSPE. If the medical school did not provide a ranking system, no attempt was made to determine an overall CR for that candidate. Alpha Omega Alpha (AOA) was given a 1 ranking and the next subsequent category a 2. If AOA was not used or specified on a particular school's MSPE, then the first category was the first breakdown specified by the school. Data were recorded according to the MSPE; therefore, a particular candidate may have an overall CR of 2/4 (second quartile) or a CR of 1/5 (AOA in a quintile system). Statistically, no distinction was made between a candidate who obtained first category of a 7-category system and first category of a 3-category system.

We used the global assessment scores on the SLOR to determine ratings on the LOR. Specifically, a global assessment score of 1 was given to outstanding, 2 for excellent, 3 for very good, and 4 for good. If more than 1 box was checked, a composite score was assigned (eg, 1.5 for outstanding and excellent). If the candidate had several SLOR global assessment scores, we averaged all of them to give the candidate a composite score. If no global assessment score was given, we did not assign a score and left it blank.

USMLE scores were used as determined from the ERAS packet. Three-digit scores for steps 1 and 2 were used. If no USMLE scores were available, this information was left blank.

Rank order list information was taken from the program's final NRMP rank list. If a candidate was unranked, one number lower than the lowest-ranked candidate was assigned. All unranked candidates received the same number.

We assigned each candidate a randomized number, and information regarding each candidate was blinded. We retrospectively examined residency applications to a single EM training program. The study was conceived after interview season was completed and the program's NRMP ROL was

Table. Spearman's rank correlation (ρ) along with associated two-tailed *P* values for information about each candidate. Significant values denoted by asterisks.

	MSPE CR	EM grade	SLOR rating	USMLE step 1	USMLE step 2	Final rank
MSPE CR						
Spearman's (ρ)		0.126	0.189	−0.572*	−0.536*	0.234*
P		0.272	0.092	0.001	0.001	0.035
EM grade						
Spearman's (ρ)	0.126		0.636*	−0.178	−0.076	0.379*
P	0.272		0.001	0.055	0.545	0.001
SLOR rating						
Spearman's (ρ)	0.189	0.635*		−0.100	−0.083	0.332*
P	0.092	0.001		0.273	0.502	0.001
USMLE step 1						
Spearman's (ρ)	−0.572*	−0.178	−0.100		0.638*	−0.253*
P	0.001	0.055	0.273		0.001	0.006
USMLE step 2						
Spearman's (ρ)	−0.536	−0.076	−0.083	0.638*		−0.348*
P	0.001	0.545	0.502	0.001		0.004
Final rank						
Spearman's (ρ)	0.234*	0.379*	0.332*	−0.253*	−0.348*	
P	0.035	0.001	0.001	0.006	0.004	

MSPE CR, Medical Student Performance Examination class rank; *EM,* emergency medicine; *SLOR,* standardized letters of recommendation; *USMLE,* US Medical License Examination.

submitted. The retrospective design was selected to minimize the potential for data extractor bias to skew results.

Data Analysis

All statistical analyses were conducted using Statistical Analysis Software (SAS) Version 9.0 (SAS Institute, Inc, Cary, NC). Spearman's rank correlation coefficient (ρ) was used to analyze and compare data. A *P* value of ≤ 0.05 was considered significant.

RESULTS

We were able to locate and examine ERAS applications for 127/129 (98.4%) of candidates. Of the 127 MSPE letters received, we were able to determine CR for 76 (59.8%) of the candidates. CRs were missing in 42/127 (33.1%) applicant packets, while for 9/127 (7.1%), the CR was reported as AOA only. CR systems utilized by medical schools included the following: thirds (21/76, 27.6%), quartiles (24/76, 31.6%), quintiles (16/76, 21.1%), 6 categories (6/76, 7.9%), 7 categories (3/76, 3.9%), and overall CR with the number of students in the class (6/76, 7.9%). Compared to CR, the following ERAS information was present more often in the applicant packets we examined: EM grades (117/127, 92.1%, $P < 0.001$), SLOR global assessment ratings (121/127, 95.3%, $P < 0.001$), and USMLE step 1 scores (125/127, 98.4%, $P < 0.001$). CR correlated positively with a candidate's final placement on the ROL; $\rho = 0.234$, $P = 0.035$ (Table).

One EM grade was noted on 117/127 of candidate applications (92.1%) with 65/127 (52%) submitting 2 or more EM grades, 6/127 (4.7%) submitting 3 or more EM grades, and 1/127 (0.79%) submitting 6 EM grades. EM grades correlated positively with a candidate's final placement on the ROL, $\rho = 0.379$, $P < 0.001$.

At least one SLOR was included in 121/127 (95.3%) of candidate applications. Two or more SLORs were submitted by 90/127 (70.9%) of candidates, while 37/127 (29.1%) candidates submitted at least 3 SLORs, 4/127 (3.1%) candidates submitted at least 4 SLORs, and 1/127 candidates submitted at least 6 SLORs. The SLOR global assessment rating correlated positively with a candidate's final placement on the ROL, $\rho = 0.332$, $P < 0.001$.

USMLE step 1 scores were submitted by 125/127 (98.4%) of candidates. Step 2 scores were submitted by 72/127 (56.7%) of candidates. Both of these correlated negatively with final placement on the ROL: USMLE step 1 scores, $\rho = -0.253$, $P = 0.006$; and USMLE step 2 scores, $\rho = -0.348$, $P = 0.004$.

DISCUSSION

Our data show positive correlations between a candidate's EM grade, SLOR global assessment, CR, and overall standing on the ROL. This supports the findings of a 1998 study by

Crane and Ferraro where EM program directors indicated that EM grades, the interview, clinical grades, other, and recommendations were felt to be most important in the selection of residents.[7] As these correlations were weakly positive, however, this may suggest other parts of the application or the application process itself may significantly affect the ultimate position of a candidate on the ROL.

The ERAS application has many components. We evaluated the relationship between EM grade, SLOR global assessment, CR, and the ultimate ROL ranking. We did not evaluate other aspects of the SLOR (ie, section on qualifications for EM), nor did we evaluate educational background, extracurricular activities, volunteerism, or research and publications. Other research in EM has shown these parts of the application to be comparatively undervalued by EM program directors and by program directors in other specialties.[7,9] That said, Hayden showed the medical school attended and the presence of distinctive factors in the application predict future success as an EM resident.[9]

The residency application process extends beyond the ERAS application. It can include informal gatherings at venues like residency fairs, visiting student audition electives, pre-interview gatherings with residents, the formal interview, return visits to programs of interest, and post-interview interactions. Survey-based research in EM and other specialties suggests that program directors of various specialties value the interview highly.[7,10–12] In addition to the interview, EM program directors value expressions of interest on the part of the candidate.[7] As none of our metrics scored a high correlation with a candidate's final rank on the ROL, our findings suggest that other factors, either from the ERAS application or the residency application process itself, may significantly affect a candidate's ultimate position on the ROL. This finding dovetails with other studies that found the selection process does not reliably predict those residents who would later succeed in residency.[13,14] Furthermore, other specialties have found nonobjective factors, such as the faculty interview, predict future residency performance.[15] Finally, a questionnaire of pediatric emergency medicine fellowship directors found the most important factor in granting an interview was recommendation from colleagues,[16] again underscoring the importance of other immeasurable factors in a candidate's application.

In addition to the positive correlation with the above, our data show a negative correlation between USMLE steps 1 and 2 scores and NRMP rank position. This is in conflict with the data of the 1998 Crane survey where EM program directors indicated that USMLE steps 1 and 2 were moderately important in the selection of applicants.[7] While perception may be that USMLE scores are important to match in an EM program, and indeed they may be used to screen applicants in many programs, in fact, these factors likely play a less important role than previously thought. This is helpful information for medical students and those who advise medical students as other factors, in particular performance on EM rotations, may have greater influence on the competitiveness of a candidate for EM residency.

Future directions for research include attempting to tease out various factors that were unmeasured in our study, such as institution-specific EM grades, SLOR author identity and role (eg, program director vs clerkship director), medical school reputation, the addition of post-interview data, and the impact of the candidate's interview performance on ultimate rank position. Furthermore, expanding this study to other institutions would broaden its external generalizability.

LIMITATIONS

Generalizability is the primary limitation of the study. Our convenience sample of candidates at a single institution may not be representative of other programs' candidates. Similarly, it may not be possible to generalize a single residency's approach to ranking with others. We studied aspects of the ERAS application and their relevance to ranking. Factors separate from the ERAS application, however, may affect a candidate's ROL placement, such as audition elective completion, personal correspondences from mentors, and interactions at social events with the residents. These were not studied. While every effort was made to extract all pertinent study data from ERAS packets, it is possible that some information may have been added after the interview date and thus excluded from analysis in this study. Furthermore, a single data abstractor who coauthored this paper extracted the data used for this study. While this is a weakness of our methodology, the data extracted was objective and therefore should have limited the bias in this study. Also, as this study used a combination of interval and ordinal data, we chose to use a Spearman's rank correlation to evaluation this data. Nonetheless, incorporating these 2 different types of datasets poses challenges statistically. Two candidate packets were missing from the study and therefore not included in data analysis. Extracting data from MSPE letters proved to be challenging, as has been noted in other publications.[17] Along these lines, we decided a 1 ranking represented either AOA in schools with that designation or first category (quartile, quintile, etc) in schools that did not use AOA. It may be difficult to draw conclusions based on these categories, as there is a wide variability in how schools rank students.

CONCLUSIONS

Our evaluation of applications for an EM residency program shows a positive correlation between a candidate's EM grade, SLOR global assessment, CR, and ROL position. Conversely, USMLE scores had a weakly negative correlation with candidates' final ranks. The fact that none of these parameters scored a strong correlation with ultimate rank position suggests that other factors in the ERAS application or the application process may influence a candidate's ultimate position on the ROL. Medical students applying for an EM

residency position should be advised that other non-USMLE factors, in particular their performance on EM rotations, may carry greater weight in determining where they end up on an ROL than previously thought.

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How Long Are Patients Willing to Wait in the Emergency Department Before Leaving Without Being Seen?

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Introduction: Our goal was to evaluate patients' threshold for waiting in an emergency department (ED) waiting room before leaving without being seen (LWBS). We analyzed whether willingness to wait was influenced by perceived illness severity, age, race, triage acuity level, or insurance status.

Methods: We conducted this survey-based study from March to July 2010 at an urban academic medical center. After triage, patients were given a multiple-choice questionnaire, designed to ascertain how long they would wait for medical care. We collected data including age, gender, race, insurance status, and triage acuity level. We looked at the association between willingness to wait and these variables, using stratified analysis and logistic regression.

Results: Of the 375 patients who were approached, 340 (91%) participated. One hundred seventy-one (51%) were willing to wait up to 2 hours before leaving, 58 (17%) would wait 2 to 8 hours, and 110 (32%) would wait indefinitely. No association was found between willingness to wait and race, gender, insurance status, or perceived symptom severity. Patients willing to wait >2 hours tended to be older than 25, have higher acuity, and prefer the study site ED.

Conclusion: Many patients have a defined, limited period that they are willing to wait for emergency care. In our study, 50% of patients were willing to wait up to 2 hours before leaving the ED without being seen. This result suggests that efforts to reduce the percentage of patients who LWBS must factor in time limits. [West J Emerg Med. 2012;13(6):463-467]

INTRODUCTION

In 2008 an estimated 124 million patients sought emergency care in the United States.[1] This number grows every year. Unfortunately, as patient volume increases, crowding and emergency department (ED) boarding of patients waiting to be admitted to the hospital increase as well.[1] This leads to prolonged wait times for many patients seeking emergency care.

The ED functions as a healthcare "safety net," providing full-time services for the general public.[2] Patients come to the ED for evaluation of emergent or urgent conditions, for after-hours medical care, or by referral from their primary physician. In striving to fulfill this vital role, EDs across the country struggle to balance demand and capacity.

Previous studies have shown that the longer patients wait, the more likely they are to leave without being seen (LWBS).[3] Studies have also shown that a significant portion of patients who LWBS are classified as needing emergent or urgent medical care.[4] The question of interest to our group was this: How long are patients willing to wait before they choose to LWBS by a health care provider?

Our study sought to evaluate patients' threshold for waiting before they choose to leave the ED waiting room without being seen by an ED provider. We also sought to determine whether willingness to wait was influenced by factors such as illness severity, age, or insurance status. Knowing the limits to which patients will wait may be useful in tailoring strategies to reduce wait times.

METHODS

Settings and Participants

Between March and July 2010, we approached patients waiting for emergency medical care in an urban academic center with approximately 46,000 adult patients annually. Patients were approached after triage but before being seen by a physician. Survey were administered between 8AM and 10PM. Patients were eligible for the study if they were between 18 and 89 years of age and had an emergency severity index (ESI) level of 3, 4, or 5. We excluded patients triaged with a higher acuity level (ESI 1 or 2) because they were taken immediately into the ED for care, without spending any time in the waiting room. We excluded prisoners, as well as patients presenting with psychiatric emergencies, including those arriving on emergency petition. Non-English-speaking patients were not excluded. Patients completed the survey by reading the questions and recording their responses on the survey form. Patients had the option of having the survey questions read to them.

Data Collection

Ten research assistants (first- and second-year medical students) collected data throughout the enrollment period. They received uniform training on survey administration and data recording. Research assistants worked in 4-hour shifts according to the following schedule: 8AM to 12PM, 2 to 6PM, and 6 to 10PM. When they approached the patient, the assistants briefly explained the purpose of the study and informed the patient that the survey was anonymous and that participating or not participating in the study would not affect care. Upon enrollment, data were collected using a standardized, multiple-choice questionnaire (Appendix). We gave the patients an information sheet describing the aims of the study and listing contact information that they could use if they had any concerns. Consent was obtained using a brief oral script. The institutional review board approvd this study .

After the survey was administered, we collected the following information: patient age, gender, race, insurance status, employment status, phone status (absence suggesting lower socioeconomic status), triage acuity level, chief complaint, and triage time using the patients' electronic health record. We also collected this information from those who declined to participate. The time of survey administration and how long the patient had waited prior to being approached were recorded as well.

Data Analysis

We compared demographic variables between respondents and non-respondents to assess the potential for selection bias. Based on the distribution of responses, we dichotomized willingness to wait into groups defined by more than 2 hours versus 2 hours or less. We assessed univariate associations of categories of predictor variables, including strata of ordinal variables versus willingness to wait more than 2 hours. Other ordinal variables were dichotomized as appropriate. We used a stratified analysis to assess for interaction between the following variables and the relationship between acuity and willingness to wait: insurance status, phone status (surrogate

Table 1. Descriptive data for 340 patients wating for emergency care.

Factor	No. (%)
Age (year)	
18-25	64 (19)
26-34	69 (20)
35-45	85 (25)
> 45	122 (36)
Female	202 (59)
Race*	
Caucasian	82 (24)
African-American	254 (75)
Other	3 (1)
Uninsured*	106 (31)
Emplyoment*	
Employed	122 (36)
Unemployed	198 (58)
Student	4 (1)
Retired	15 (4)
Phone contact listed	317 (93)
Waited ≥ 30 minutes prior to survey	179 (53)
Triage acuity level†	
Level 3	193 (57)
Level 4	120 (36)
Level 5	23 (7)
Severity of symptoms	
Mild	55 (16)
Medium	125 (37)
Severe	160 (47)
Concern for symptoms	
Little	36 (11)
Somewhat	96 (28)
High	208 (61)
Prior visits to this ED this year*	
0	173 (51)
1-2	98 (29)
3-5	45 (13)
≥ 6	23 (7)
Need for this ED*	
Any ED will do for today's problem	157 (46)
Prefer this ED for today's problem	114 (35)
Need this ED for today's problem	68 (20)

ED, emergency department
*Data not available for one patient
†Emergency Severity Index triage system, missing data for 4 patients

Table 2. Univariate associations with willingness to wait > 2 hours. Includes data from 339 patients who indicates a duration they were willing to wait.

Factor	Fraction of those with factor willing	Fraction of those without factor willing	% Difference (95% CI), p-value
Age (year)			
18–25	21/64 (33%)	147/275 (53%)	(-)21 [(-)7–(-)35], 0.003
26–34	40/69 (58%)	128/270 (47%)	11 [(-)3–(+)25], 0.09
35–45	46/84 (55%)	122/255 (48%)	7 [(-)6–(+)20], 0.3
> 45	61/122 (50%)	107/217 (49%)	1 [(-)11–(+)12], 0.9
Female gender	96/202 (48%)	72/137 (53%)	(-)5 [(-)16–(+)6], 0.4
Minority race/ethnicity*	128/257 (50%)	40/82 (49%)	1 [(-)12–(+)14], 0.9
Uninsured†	53/106 (50%)	114/232 (49%)	1 [(-)11–(+)13], 0.9
Unemployed†	114/212 (54%)	53/126 (42%)	12 [0–23], 0.04
Symptoms*			
Severe discomfort	87/159 (55%)	81/180 (45%)	10 [(-)2–(+)21], 0.08
High concern for cause	106/207 (51%)	62/132 (47%)	4 [(-)7–(+)16], 0.5
Higher triage acuity‡	109/192 (57%)	49/143 (34%)	23 [11–34], 0.006
ED preference†			
Need this ED	37/68 (54%)	131/270 (49%)	6 [(-)8–(+)20], 0.4
Prefer or need this ED	106/182 (58%)	62/156 (40%)	19 [7–30] , 0.0007
Wait ≥ 30 min prior to survey*	101/178 (57%)	67/161 (42%)	15 [4–26], 0.007

ED, emergency department
*Response not provided by one patient
†Response not provided by two patients
‡Emergency severity index level 3 (versus 4 or 5), triage level not recorded for 4 patients

Table 3. Adjusted odds ratios for willingness to wait < 2 hours or more.

Factor	Adjusted odds ratios [95% CI], p-value
Age > 25 years	2.5 (1.4–4.5), 0.003
High acuity (ESI 3)	1.7 (1.0–2.6), 0.03
Wait 30 minutes	1.6 (1.0–2.6), 0.03
Preference for this emergency department	1.9 (1.2–3.0), 0.004

ESI, emergency severity index

Table 4. Potential response to excessive waiting time.

Factor	No. (%)
See a primary care MD next day	28 (8)
Come back to this ED next day	33 (10)
Go to another ED same day	83 (24)
Would not see an MD for complaint	12 (4)
Would wait indefinitely	181 (53)
No answer	3 (1)

ED, emergency department

for lower socioeconomic status), wait thus far, age category, and need for this ED. We defined potential interaction as a Broselow-Day significance < 0.05.

We prepared a multivariable logistic model that included acuity and any other variables associated with willingness to wait more than 2 hours at significance < 0.1. We removed variables from the model as long as they did not change the model coefficient for acuity by more than 10%, did not decrease the precision for this estimate by more than 10%, or had significant adjusted associations with willingness to wait at $P < 0.05$.

RESULTS

Of the 375 patients eligible to participate, 340 (91%) consented to complete the questionnaire. One participant answered all of the questions except how long he/she was willing to wait. Characteristics of the study population are shown in Table 1. Figure 1 indicates the distribution of stated willingness to wait times, which was bimodal, with 171 (51%) indicating willingness to wait up to 2 hours and 110 (32%) indicating they would be willing to wait indefinitely. The remaining 58 (17%) were willing to wait various times between 2 and 8 hours before leaving. The 35 patients who refused to participate were similar to participants in age distribution, gender, race, insurance status, phone status, acuity level, and wait time prior to contact.

Associations between predictor variables and willingness to wait are shown in Tables 2 and 3. Among social and demographic factors, including insurance status, race, gender, and age; only age greater than 25 years was independently

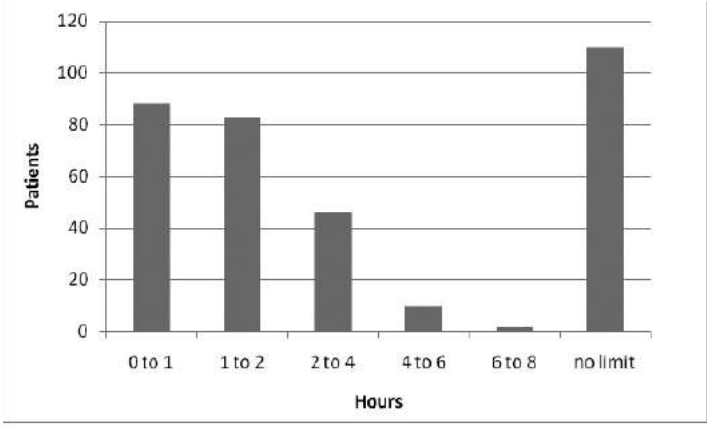

Figure 1. Distribution of patients and the number of hours willing to wait to be seen by medical care providers in the emergency department.

associated with a willingness to wait longer to be seen. Although patients' subjective concern for symptoms and severity of symptoms were not statistically associated with willingness to wait, patients with a higher acuity, as indicated by the triage-assigned acuity (ESI level 3), were more willing to wait, by both crude and adjusted analyses. Preference for the study site ED also predicted increased willingness to wait.

Table 4 displays patients' responses when asked what they would do next if they decided to leave the waiting room without being seen. The vast majority of patients, 96%, indicated that they would seek care for their health concern elsewhere.

LIMITATIONS

Our major limitation is that we did not survey patients from 10PM to 8AM. Patients who present during the overnight hours may be more inclined to wait because they have limited alternative care options. In addition, our study population consisted of ED patients presenting to an inner city, academic, tertiary care referral center. The study group was predominantly African-American. Over 50% of patients surveyed preferred our ED. These patients may have been more inclined to wait because of the inconvenience of going to another ED or because of their familiarity with the study site ED. Patients' willingness to wait may be different in a community, non-academic setting.

Half of the participants revealed that 2 hours was the limit they would wait. We did not retrospectively review to see how many of these patients stayed true to their initial survey response. There might be a difference between how long patients say they are willing to wait and how long they actually wait. Comparing their initial response with the actual duration of time they decided to stay would have provided additional information regarding willingness to wait. A final limitation involves the exclusion of ESI 1 and 2 patients. Thus, a large group of patients might be excluded from the applicability of the study's findings.

DISCUSSION

Much has been written in the past decade about the large number of patients leaving the nation's EDs without being seen by a physician.[2,3] Wait times for patients desiring to be seen by an emergency physician can stretch for hours. Additionally, the annual census of the nation's EDs continues to be well above 100 million patient visits.

At a certain time, patients may decide that waiting longer to see a physician in the ED waiting room is beyond what they can tolerate.[5-7] Our study evaluated the length of time that patients stated they were willing to wait before leaving without being seen and what variables may be associated with a willingness to wait longer. Of the 340 patients who participated, 171 (51%) had a threshold of 2 hours. Among demographic factors, only age above 25 years was independently associated with a willingness to wait more than 2 hours. Previous studies looking at characteristics of patients who left without being seen had also found that younger patients have a higher uncompleted visit rate compared with older individuals.[4,8] Those studies made no mention of when the patients left or how long they waited.

Our study found that patients whose conditions were deemed to be of higher acuity by the triage nurse were more willing to wait beyond 2 hours. This disconnect might have been due in part to conveyance to the patient by the triage nurse, purposeful or not, as to the level of severity of his/her presenting illness. This might have influenced the patient's decision. Interestingly, we did not find the same willingness to wait among patients who classified themselves as being in severe discomfort and/or having a high level of concern for their symptoms.

A preference for the study site ED also was associated with a willingness to wait beyond 2 hours. Although we did not explore the specific reasons for this preference when the study was conducted, we feel that the effect of ED preference on a patient's willingness to wait is plausible and not surprising. Every ED has its core group of patients who return for treatment of varying illnesses. For reasons of convenience (access, location) and/or comfort with the care they have received in the past at the facility, a preference develops and is likely to mitigate perceived or actual wait times. Additionally, given that our study site is a tertiary care facility, some patients may require specialty care at our hospital and may feel they cannot receive the same care at other hospital EDs.

An encouraging finding in our study was that in response to excessive wait times, 96% of our cohort would continue to seek the services of a healthcare provider for their illness. They could find this help by seeing their primary care physician the following day, going to another ED that same day, returning to the same ED later in the day, or just waiting indefinitely at the survey site ED.

CONCLUSION

Decreasing the number of patients who LWBS is an

important goal for hospitals across the country. The ED has an important role in the country's healthcare system, providing emergency services as well as care for patients with urgent concerns after hours or with limited access to medical care. Half of the patients we surveyed were willing to wait as long as 2 hours before leaving the ED waiting room without being seen. This finding could be used as a guide to support capacity management decisions, recognizing that the risks and lost revenue from patients leaving without being seen are defined by time constraints. Despite lengthy wait times, it is reassuring that few patients presenting to the ED for evaluation will forego the services of a physician completely. Patients seem to be intent on receiving care for their particular illness, but many have a defined, limited period that they are willing to wait in the ED waiting room.

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Family History Is a Predictor for Appendicitis in Adults in the Emergency Department

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Introduction: A family history of appendicitis has been reported to increase the likelihood of the diagnosis in children and in a retrospective study of adults. We compare positive family history with the diagnosis of acute appendicitis in a prospective sample of adults.

Methods: We conducted a prospective observational study of a convenience sample of 428 patients. We compared patients with surgically proven appendicitis to a group without appendicitis. The latter were further grouped by their presenting symptoms: those presenting with a chief complaint of abdominal pain and those with other chief complaints. Participants answered questions regarding their family history of appendicitis. Family history was then compared for the appendicitis group versus the nonappendicitis group as a whole, and then versus the subgroup of patients without appendicitis but with abdominal pain. The primary analysis was a χ^2 test of proportions and the calculation of odds ratio (OR) for the relationship between final diagnosis of appendicitis and family history.

Results: Of 428 patients enrolled, 116 had appendicitis. Of those with other diagnoses, 158 had abdominal pain and 154 had other complaints. Of all patients with appendicitis, 37.9% (confidence interval [CI] = 29.1–46.8) had positive family history. Of those without appendicitis, 23.7% (CI = 19.0–28.4) had positive family history. In the subgroup without appendicitis but with abdominal pain, 25.9% (CI = 19.1–32.8) had positive family history. Both comparisons were significant ($P = 0.003$; OR = 1.97; 95% CI = 1.2–3.1; and $P = 0.034$; OR = 1.74; 95% CI = 1.04–2.9, respectively). By multivariate logistic regression analysis across the full sample, family history was a significant independent predictor ($P = 0.011$; OR = 1.883) of appendicitis.

Conclusion: Adults presenting to the emergency department with a known family history of appendicitis are more likely to have this disease than those without. [West J Emerg Med. 2012;13(6):468–471.]

INTRODUCTION

Background

Appendicitis is a common cause of acute abdominal pain, for which surgical intervention is required, and has been recognized as one of the most common causes of the acute abdomen worldwide. In the United States, there are approximately 250,000 cases of appendicitis per year, with an incidence of 1.1 cases per 1,000 people per year.[1] The diagnosis hinges on an index of suspicion based on a careful history and physical examination. "Textbook" cases often require no imaging studies and go straight to the operating room; however, many patients do not present with classic symptoms and have

nonspecific features, including indigestion, a sense of not feeling well, or nonlocalized pain. The diagnosis of appendicitis can be especially challenging in women and patients older than 60 years owing to the presence of other possible pelvic inflammatory causes in the former and often delayed presentation in the latter.[2]

Family history is an important factor in the risk stratification of various diseases. Studies done in the late 1970s and 1980s showed a correlation between a positive family history and appendicitis in children.[3,4] In a more recent study, Gauderer et al[5] performed a prospective case-control study assessing the importance of family history in a pediatric population. In that study children with appendicitis were twice as likely to have a positive family history when compared to controls with right lower quadrant pain that had a diagnosis other than appendicitis. The authors also found that children with appendicitis were 3 times as likely to have a positive family history as controls without abdominal pain. A recent chart review of 2,670 patients reported that a family history of appendicitis increased the likelihood of disease by threefold.[6]

Importance

To date there is no prospective case control study in adults examining the ability of family history to predict appendicitis in adults.

If it can be shown that family history increases the likelihood for the disease in adults as well as children, it will enhance the ability of the clinician to make the diagnosis on clinical grounds more confidently. This is important since patients for whom the diagnosis cannot be made or ruled out with confidence require further diagnostic testing, usually by computed tomography (CT), with increased costs in time, money, and radiation exposure. The risks of missing the diagnosis with attendant medical and surgical complications are well known. Better risk stratification may ultimately reduce the number of scans performed and improve clinical accuracy.

Objective

The objective of our study was to determine if family history is a risk factor for appendicitis in the adult population and thus, if it can be used as a predictor for the clinical diagnosis of appendicitis.

METHODS

Study Design

We enrolled a convenience sample of patients presenting to the emergency department (ED) into a prospective case-control study. We recruited equivalent numbers of subjects (1:1:1) in case groups and control groups 1 and 2. Sample sizes of 115 in the case group and 115 in the each of the control groups (230 in the combined nonappendicitis group) were calculated to achieve 80% power, to detect a difference of 16% between the null hypothesis, stating that positive family history is present in all groups at a proportion of 14%, and the alternative hypothesis, stating that the proportion in the case group is 30% (an odds ratio [OR] of 2.63), by using a 2-sided χ^2 test with continuity correction and with a significance level of 0.05. The study was approved by the institutional review board.

Setting and Population

The setting was a tertiary urban teaching referral center. Patients were enrolled on a convenience basis, both with and without appendicitis, and based on availability of a researcher. Cases and controls were not individually matched. Patients with a diagnosis of appendicitis were identified from hospital admission records by admitting diagnosis, which was confirmed at surgery, and the patients were enrolled during the same admission. Patients were excluded if they were younger than 18 years, adopted, unaware of their family history, critically ill, or had a prior appendectomy.

Study Protocol

All patients were interviewed in person by using a standard data collection form. They were asked specifically whether or not the patient's parents, brothers, sisters, grandparents, or children had had appendicitis. Gender, age, and ethnicity were also noted. Data were statistically analyzed with SPSS 14.0 (IBM, Armonk, New York).

Measurements

Patients were categorized according to final diagnosis: those with surgically proven appendicitis and those who were otherwise diagnosed. Patients in the latter group were further categorized by their presenting symptoms: those presenting with a chief complaint of abdominal pain and those with other symptoms. A patient was considered to have a positive family history if any first-degree relative or grandparent was known to have had appendicitis. If the patient did not know of a diagnosis of appendicitis in the family, this was taken to be a negative family history.

Data Analysis

The primary analysis was a χ^2 test of proportions and the calculation of OR and 95% confidence intervals (95% CI) for the relationship between final diagnosis of appendicitis and family history. We did this to answer the general question of whether patients with appendicitis are more likely to have a positive family history than patients with all other diagnoses. We also, following a previously published methodology, wanted to try and answer a subsidiary question: Are patients with appendicitis more likely to have a positive family history than those patients presenting *specifically with abdominal pain* but eventually found NOT to have appendicitis? We therefore further compared the appendicitis group to the subgroup of patients without appendicitis who presented with abdominal pain. Logistic regression was used to examine family history in the prediction of the final diagnosis, while controlling for demographic factors.

Table. Demographic characteristics.

Characteristic	Case group: appendicitis (n = 116)	Control 1: chief complaint of abdominal pain (n = 158)	Control 2: chief complaint other than abdominal pain (n = 154)
Age			
≤30	36	39	26
31–45	35	54	36
46–60	28	37	40
≥61	17	28	52
Gender			
Male	62	53	67
Female	54	105	87
Race			
White	83	92	97
Black	7	29	25
Hispanic	23	35	31

Outcome Measure

We measured whether the proportion of patients having appendicitis and a positive family history was larger than the proportion of patients in control groups with positive family history.

RESULTS

We enrolled 428 patients, 116 of whom had appendicitis and 312 who did not. Among the nonappendicitis group, a subgroup of 158 patients presented with abdominal pain (from another diagnosis) The demographic comparison among the groups is shown in the Table. Significant differences were found in age ($P = 0.04$), gender ($P = 0.005$), and race ($P = 0.04$), with younger, male, and white patients more likely to be in the appendicitis group.

Of the 116 patients with appendicitis, 44 (37.9%; CI = 29.1–46.8) had a positive family history. Of all 312 patients without appendicitis, 74 (23.7%; CI = 19.0–28.4) had a positive family history. In the subgroup of 158 patients who presented with abdominal pain but did not have appendicitis, 41 (25.9%; CI = 19.1–32.8) had positive family history. Both comparisons were significant ($P = 0.003$; OR = 1.97; 95% CI = 1.2–3.1; and $P = 0.034$; OR = 1.74; 95% CI = 1.04–2.9, respectively).

Given the presence of significant relationships between diagnosis and demographics in the current data set, we ran a multivariate logistic regression analysis. Family history was a significant independent predictor of a diagnosis of appendicitis among the full sample of patients presenting to the ED ($P = 0.011$; OR = 1.883; 95% CI = −1.16 to 3.06). When those with appendicitis are compared only to the smaller sample without appendicitis but presenting with abdominal pain, the result, while similar (OR = 1.612; 95% CI = 0.932–2.788), does not reach statistical significance.

DISCUSSION

In our study, patients who presented to the ED with appendicitis were more likely to have a positive family history than those presenting with other diagnoses, whether or not they had abdominal pain. This difference was statistically significant in all comparisons except when using logistic regression analysis and limiting the comparison to those presenting with abdominal pain. The OR in this comparison was similar to the odds ration when comparing all patients (OR, 1.61 vs OR, 1.88). When using a smaller sample, it is more difficult to show statistical significance; in light of the rest of our results, this is likely to account for the discrepancy.

A previous retrospective study of patients taken to the operating room for appendectomy, which compared those having appendicitis to those without, showed an increased incidence of family history in patients with appendicitis versus those without, with a greater difference in those younger than 20 years, for which family history was positive in 45% of cases with appendicitis versus 17% of cases without.[7] This study did not apply statistical methodology to its results and was potentially subject to various biases, including work-up bias; however, it was done in an area of India where clinical diagnosis is the sole determinant for surgery, and its findings are consistent with ours.

On its own, the increased likelihood of appendicitis among those with a positive family history is not enough to diagnose or rule out the disease. However, this element—the history of the present illness—will help inform the pretest likelihood of a given patient's having appendicitis, as the clinician determines the threshold for further testing. The concept of constructing a pretest likelihood from various relatively nonspecific risk factors to determine further testing is well known from the diagnostic algorithms for diseases such as pulmonary embolism and deep vein thrombosis.[8,9] We think that taken in the context of the rest of the clinical signs and symptoms, a family history will add modestly to the clinical *gestalt* of the patient when there is diagnostic uncertainty, especially when CT is not routinely unavailable.

LIMITATIONS

Our study was a convenience sample of patients who presented to a single emergency department. Given this sampling approach, it is not known whether the resulting sample was representative of all adults presenting to ED or whether factors, such as day of week or time of day, or indeed the specific population using this ED, led to any bias. Thus, the resulting sample could be nonrepresentative, and this limits the external validity of the conclusions. The possibility that there were any confounding differences between the groups could limit the internal validity as well. Multivariate regression was used to ameliorate the possible limitations to internal validity, but it is not possible to know how completely this was accomplished. Data were collected during a 4-year period owing to the availability of staff to enroll patients in our study. Recall bias is

another potential limitation of our study. Our results relied solely on the patient's knowledge of their family history. We did not verify whether or not their recall was correct. One may ask whether our study suffers from "work-up bias." Work-up bias occurs whenever a test is performed and a "gold standard" (reference) validation is not performed for each patient, and accuracy of the test is reported for only patients with reference validation. This is particularly apt to occur when the gold standard involves an invasive procedure.[10] In our study, if one takes the gold standard as surgery for probable appendicitis, it is true that not all our patients underwent that procedure in order to definitively rule in (or rule out) the diagnosis. If there were a large group within our sample that had appendicitis but were not taken to surgery, then this might be a confounding factor. However, it is unlikely that our diagnostic test was used to determine which patients would go to surgery. As one source put it, ". . . we have observed *lack* of work-up bias only in settings in which a surgeon does not believe in the test or ignores it for purposes of decision making . . ."[10]

CONCLUSION

This study suggests that, as with other disease processes, knowledge of family history of appendicitis can assist the physician in determining the likelihood of the diagnosis. Given the difficulty in diagnosing appendicitis clinically, this information could be useful in the ED setting.

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Current Trends in the Management of Difficult Urinary Catheterizations

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Routine urinary catheter placement may cause trauma and poses a risk of infection. Male catheterization, in particular, can be difficult, especially in patients with enlarged prostate glands or other potentially obstructive conditions in the lower urinary tract. Solutions to problematic urinary catheterization are not well known and when difficult catheterization occurs, the risk of failed catheterization and concomitant complications increase. Repeated and unsuccessful attempts at urinary catheterization induce stress and pain for the patient, injury to the urethra, potential urethral stricture requiring surgical reconstruction, and problematic subsequent catheterization. Improper insertion of catheters also can significantly increase healthcare costs due to added days of hospitalization, increased interventions, and increased complexity of follow-up evaluations. Improved techniques for catheter placement are essential for all healthcare personnel involved in the management of the patient with acute urinary retention, including attending emergency physicians who often are the first physicians to encounter such patients. Best practice methods for blind catheter placement are summarized in this review. In addition, for progressive clinical practice, an algorithm for the management of difficult urinary catheterizations that incorporates technology enabling direct visualization of the urethra during catheter insertion is presented. This algorithm will aid healthcare personnel in decision making and has the potential to improve quality of care of patients. [West J Emerg Med. 2012;13(6):472–478.]

INTRODUCTION

Acute urinary retention (AUR) and other genitourinary conditions often lead to difficult catheterizations. Male catheterization, in particular, can be difficult, especially in patients with enlarged prostate glands or other potentially obstructive conditions in the lower urinary tract.[1] Solutions to problematic urinary catheterization are not well known and when difficult catheterization occurs, the risk of failed catheterization and concomitant complications increase. Even routine urinary catheter placement may cause trauma and poses a risk of infection.[1,2] Methods to reduce the incidence of infection are particularly relevant since the Centers of Medicare and Medicaid Services (CMS) under rule CMS-1533-FC no longer reimburse for catheter-associated urinary tract infections.[3] The National Quality Forum, a nonprofit organization that develops national priorities and goals for performance improvement to enhance the quality of healthcare, estimated that 17% to 69% of catheter-associated urinary tract infections may be prevented with recommended infection control measures.[4] Such measures could result in up to 38,000 preventable infections and 9,000 preventable deaths related to these infections per year.[4] Repeated and unsuccessful attempts at blind urinary catheterization result in stress and pain for the patient, injury to the urethra, potential urethral stricture requiring surgical reconstruction, and problematic subsequent catheterization. Improper insertion of catheters also can significantly increase healthcare costs due to added days of hospitalization, increased interventions, and increased complexity of follow-up evaluations.[5] Therefore, all healthcare personnel who perform urinary catheterizations should be well trained in techniques specific to managing difficult catheterizations.

Much of the work of emergency physicians involves preparing for an action-specific intervention for an illness or particular scenario. Whether airway intervention, treatment of the coagulopathic trauma patient, or managing a myocardial infarction, established protocols guide how each case should be approached. However, beyond attempting to place a Foley catheter or contacting an urologist to intervene, alternative pathways for promptly managing the complicated catheterization patient are limited. For AUR, it is not uncommon for the nursing staff to attempt placement of a urinary catheter before the emergency physician is contacted of the patient's presence. If unsuccessful, repeated attempts with the same catheter may occur, catheterization with a catheter in a different size (typically larger) may be attempted, another catheter type may be used, or another healthcare worker may attempt the process. Such multiple attempts frequently result in injury to the urothelium, which is only 3 to 4 cell layers in thickness. In this worse-case scenario, the emergency physician is presented with a patient who has experienced multiple catheterization attempts, resulting in an iatrogenic injury. Repeated blind attempts at catheterization should be avoided to prevent escalation of a complex injury from what many consider to be a minor procedure.

Little is taught about urinary catheter placement during residency and it generally is allocated to the lowest level of training, often the medical student on the trauma service. While most emergency physicians probably never considered placement of a Foley catheter to be difficult or dangerous, what options are available when the attempt fails and the patient needs prompt relief? Another attempt may not be the best choice. Current emergency medicine teaching does not offer much guidance for managing difficult catheterizations, with urology consultation recommended when a transurethral catheter does not provide adequate bladder drainage.[6] Knowledge on this topic remains sparse in both emergency medicine and nursing specialties, and recommendations seldom are supported by evidence-based research. Best practice methods for blind urinary catheter placement, based on the literature and personal experience, will be summarized in this review. In addition, for progressive clinical practice, an algorithm for the management of difficult urinary catheterizations that incorporates new technology enabling direct visualization of the urethra during catheter insertion will be presented. This algorithm will aid healthcare personnel in decision making and has the potential to improve quality of care of patients.

INITIAL CATHETERIZATION

Initial management of AUR involves prompt bladder decompression, for which there are no uniform guidelines. An initial attempt at transurethral catheterization to establish drainage is appropriate for most patients. Urethral injury, either confirmed or suspected, is an absolute contraindication to urethral catheterization.[1,2] Relative contraindications include urethral stricture, recent urethral or bladder surgery, and a combative or uncooperative patient.[2] Although the classic teaching triad consisting of meatal blood, distended urinary bladder with the inability to pass urine, and a high-riding prostate, raises the suspicion of urethral injury, it is infrequently reported in the medical literature and its absence should not exclude the diagnosis.[7,8] A recent investigation of 46 patients by Shlamovitz and McCullough[8] demonstrated that no patients with urethral or bladder injuries had a high-riding prostate, which is a clinical finding that continues to be overemphasized despite its low sensitivity for the presence of lower urinary tract injury. Retrograde urethrography is the preferred diagnostic technique to investigate injury to the urethra.[7,9–11]

The main types of urinary catheters used today include the Foley (self-retaining balloon), Robinson (no balloon), Coudé (curved-tip Foley with or without balloon), irrigation (3 ports), and the external Texas catheter. Size is referred to by using the French (Fr) scale (circumference in mm), in which 1 Fr equals 0.33 mm in diameter.[12] An easy method of conversion between scales is to remember that each millimeter in diameter is approximately 3 Fr; therefore, an 18-Fr catheter is about 6 mm in diameter.[12] Initial catheterizations most commonly are performed using a Foley catheter. The adult male urethra is typically 30 Fr and selection of a 16- or 18-Fr catheter is appropriate for most men.[2,12,13] Smaller catheters (12 to 14 Fr) may be required for patients with urethral stricture, whereas patients with prostate enlargement may benefit from larger sizes (20 to 24 Fr) to avoid kinking as the catheter traverses the prostatic urethra.[2] Larger catheters with irrigation capacity should be selected for patients with gross hematuria to prevent obstruction of the lumen by blood clots and subsequent urinary retention.[2]

Women infrequently pose a challenge for urinary catheter placement. Most issues are related to vaginal atrophy or retraction of the urethral meatus into the vagina.[14] In females, shorter catheters may be used for one-time catheterizations and may prevent difficult catheterizations.[12,15]

Proper placement technique is critical, as failed attempts at catheterization may lead to iatrogenic injury. Forcing a catheter past the point of resistance can cause injuries ranging from a mucosal tear to more serious false passages (perforations), which are associated with infection, urethral stricture, and subsequent surgical management.[1,13,15,16] In turn, urethral stricture may make future catheterizations problematic.[1] The most common injury sites are the posterior and bulbous urethra.[5] The most frequent injuries are false passages created by forceful catheterization, as well as mucosal and submucosal tissue tears caused by balloon inflation in an improper position in the urethra.[5,14] Bleeding typically is the first sign that an injury has occurred. Besides making manual catheterization more difficult, bleeding also complicates subsequent endoscopic procedures that may be required.

Urology consultations for catheter placement often occur

Table 1. Tips for successful blind urinary catheterization.

Tip	Benefit
Injection of 10 to 15 mL water-soluble lubricant-anesthetic into the urethral meatus with placement of a urethral clamp 3 to 5 minutes before catheter insertion.	· Lubricant-anesthetic anesthetizes mucosa and distends urethra to facilitate catheterization. · Use of urethral clamp prevents lubricant-anesthetic from leaking out of urethra.
Elongate penis in upright position at ≈ 60° angle in line with normal anatomic curve without compressing urethra.	· Facilitates catheter insertion and passage.
Ask patient to take slow, deep breaths to help relax as catheter approaches the bulbomembranous urethra.	· Assists navigation through external sphincter.
Always complete catheter insertion to the Y hub.	· Urine easily can drain while catheter tip is placed anywhere beyond membranous urethra/external sphincter, most commonly in the posterior urethra. · Initial return of urine often is mistaken as indication that catheter reached the bladder. · Insertion to the bifurcation of the Y ensures catheter has reached bladder. · Premature balloon inflation, when return is present but catheter is not inserted into the bifurcation, will damage the urethra and can cause significant hematuria.
In uncircumsized patients, reduce foreskin to anatomic position after procedure is complete.	· Prevents paraphimosis, which can lead to ischemia of the glans penis.
If first attempt is unsuccessful, repeat with 18-Fr Coudé catheter, followed by an attempt with a 12-Fr silicone catheter, if necessary.[14]	· The Coudé offers advantages to negotiate acute angle of prostatic urethra. Ideal for cases of BPH, incorrect technique, and anxious patient. · The 12-Fr provides a smaller-caliber catheter, with the silicone material adding stiffness and limiting coiling. Ideal for advancing through medium-sized strictures and bladder neck contractures.

BPH, benign prostatic hypertrophy.

when there is no organic pathology, but instead, when improper catheter placement has caused urethral injuries in patients with normal problematic anatomy.[14] After analysis of data at 2 different institutions, Villanueva and Hemstreet III[14] reported that 41% and 54% of patients requiring urologic consultation for difficult urinary catheterization were readily catheterized with an 18-Fr Coudé catheter. In the authors' experience, the most common cause of difficult urinary catheterizations was an incorrect technique or benign prostatic hypertrophy (BPH) rather than urethral stricture disease.[14] Before ordering a urology consultation or advancing to more complex techniques, the tips outlined in Table 1 may be beneficial in increasing the likelihood of successful passage of a urinary catheter without causing injury.[1,2,5,12,14,17,18] Randomized controlled trials have established that the use of topical anesthetic gel reduces the pain associated with urethral catheterization in both men and women,[19,20] although the timing of delayed versus immediate catheterization may be debatable.[21] To prevent extrusion of the anesthetic gel, place a penile clamp below the glans of the penis to gently compress the pendulous urethra. Performing these steps early in the setup for catheterization allows sufficient time to provide adequate analgesia.

Suprapubic catheterization may be needed after failed attempts at transurethral catheterization or if there is pelvic or urethral trauma.[11,16,22–24] It typically is indicated when there is a tight stricture in a patient who is a good candidate for urethroplasty or when a glide wire cannot be secured in the bladder owing to a completely obliterated urethra.[14] Coagulopathy and active bladder cancer are contraindications.[14] Interventional radiologists or urologists typically perform this procedure, but it also may be performed by an emergency physician if these specialists are not available. Depending on community practice style and availability of the specialists, percutaneous suprapubic catheterization with ultrasound guidance may be easily performed at the bedside.[11,14,22,25]

DIFFICULT CATHETERIZATION ALGORITHM

Urologic history, along with clinical observations from initial unsuccessful urethral manipulation, often provides insight into the problem that is preventing catheterization.[12] For example, patients with a history of open prostatectomy may have a bladder neck contracture, whereas patients with a history of gonococcal urethritis likely will have a pendulous urethral stricture.[12] A difficult catheterization scenario in the male patient is illustrated through endoscopy in Figure 1. When difficult catheterizations are encountered, the solutions provided in Table 2 may assist in obtaining successful passage without causing injury.[2,5,12,14,18]

Since ancient Greece, urinary catheters have been placed blindly.[1,15] Urinary catheterization is considered an essential

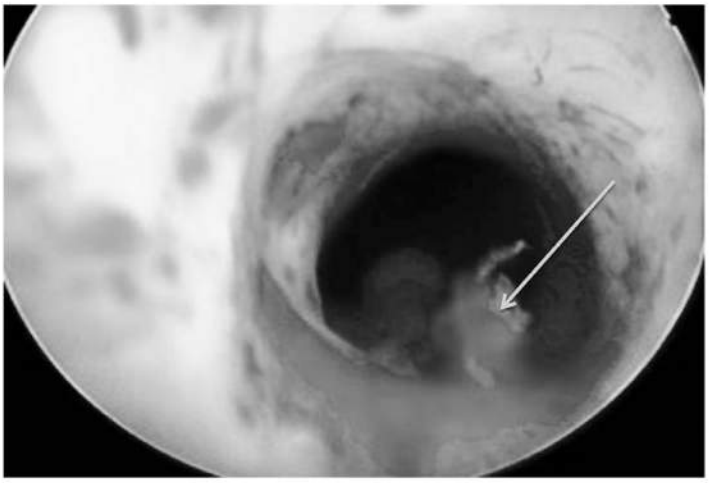

Figure 1. Flexible cystoscopy photograph with arrow indicating a urethral mucosal tear.

skill for physicians and, in the past, required surgery when blinded attempts failed. The last major advancement in urinary catheterization occurred when rubber catheters were introduced in the 18th century.[15] New technology now is available that enables direct visualization of the urethra while passing a catheter. One such system (DirectVision System; PercuVision, Westerville, Ohio) consists of a microendoscope that inserts into 1 lumen of a 3-way/trilumen Foley catheter. The microendoscope is connected to a camera and LED (light emitting diode), transporting light to the catheter tip and an image back to the LCD (liquid crystal display) monitor for real-time visualization of the urethra during catheter placement. Irrigation is used during catheter placement to assist in expanding the urethra, activating the lubricious coating of the catheter, and preventing debris from covering the lens at the distal tip of the catheter. A curved (Coudé) tip assists in navigating the normal S-shaped curve of the bulbous urethra. The procedure may be performed by any health professional (surgeon, physician, nurse, or allied health staff) trained to insert Foley catheters. The minimal training required to learn

Table 2. Possible solutions for commonly encountered difficulties during blind urinary catheterization.

Difficulty	Possible solution
Inability to locate urethral meatus in females.	· Use vaginal speculum to aid in locating urethra. · Use vaginally placed finger as a guide, with cephalad catheter placement.
Pain or resistance early in placement, while in penile or bulbomembranous urethra.	· False passage is likely—stop procedure. Forceful passage likely will be unsuccessful and can cause urethral trauma. · Refer to algorithm for solution.
Inability to pass S-shaped bulbous urethra curve.	· Stop procedure. · Reattempt catheterization with a latex Coudé catheter.
Resistance during passage through external sphincter.	· Stop procedure, ask patient to cough or relax urinary sphincter muscles as if going to void, and reattempt passage. · If substantial pain or resistance is encountered, bladder neck contracture is possible—stop procedure. Forceful advancement likely will be unsuccessful and can cause urethral trauma. · Reattempt catheterization with a latex Coudé catheter, starting at 12 Fr, with tip positioned upward. · Coudé tip may allow negotiation of the lip, which often is present at 6-o'clock bladder neck position in men with bladder neck contractures. · Maintain curved tip in same position during passage, with 12-o'clock position (curved tip pointing up) marked at the connector end of the catheter. · If catheter cannot be passed, pull back 2 to 3 cm, rotate it to 9-o'clock position, and reinsert. · If resistance still is encountered, pull back 2 to 3 cm, rotate it to 3-o'clock position, and reinsert.
Urine does not drain after full-length catheter insertion.	· Wait 5 minutes for drainage to occur. · Palpating bladder for fullness or flushing catheter with saline may force urine to open catheter tip holes, which may be blocked with gel. · If urine does not flow, do not inflate balloon, as this may cause trauma if catheter was not in bladder. · Confirm catheter insertion to the Y hub.
Pain during balloon inflation.	· Immediately stop inflation, as tip of catheter still may be in urethra. · Confirm catheter insertion to the Y hub.

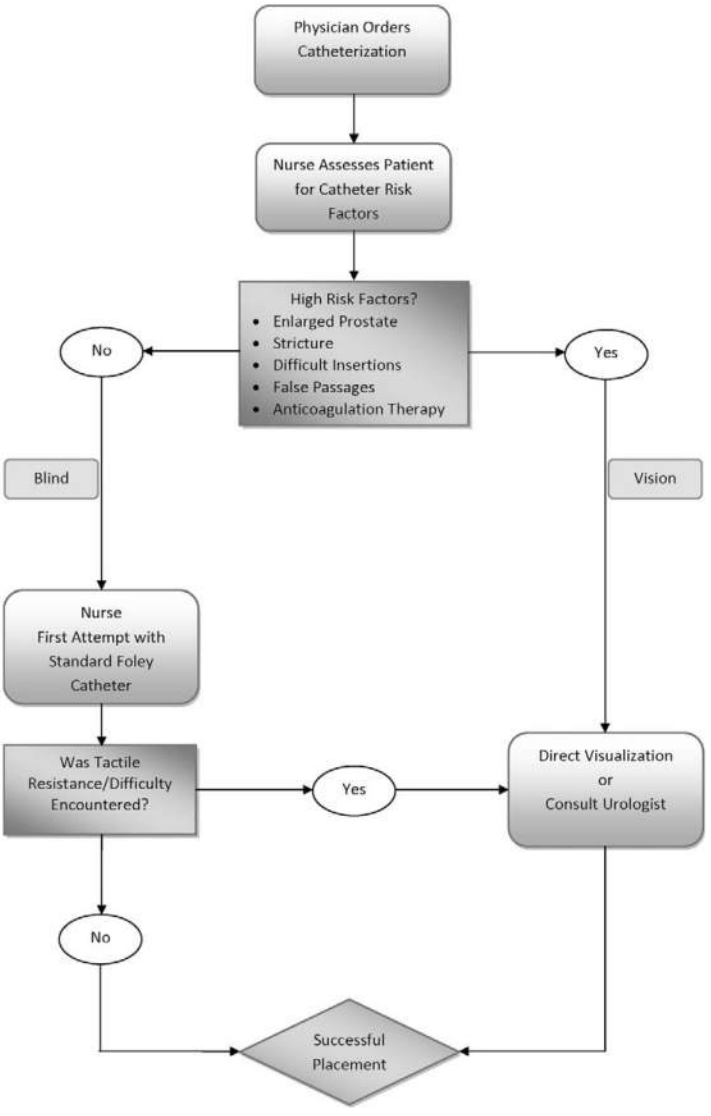

Figure 2. Algorithm for guiding decision making during difficult urinary catheterization cases.

equipment may be incorporated into existing nursing or residency training.[26]

Direct visualization of the urethra enables identification of the source of resistance, obstruction, or other complication preventing blind catheter placement. Once the source of resistance is identified, a decision whether to proceed with placement under direct visualization or to stop the attempt and seek assistance may be made. In many cases, visualization may allow the source to be circumnavigated and the catheter may be successfully placed. The ultimate goal of such technology is to limit the escalation of procedures required for a successful catheterization. Although not recommended for routine catheterization, it is best used in several high-risk clinical scenarios. The algorithm depicted in Figure 2 incorporates these scenarios and may be useful in navigating difficult catheterizations.

A pilot study examined the feasibility and safety of male urinary catheterization performed by trained emergency nursing personnel using a specific visually guided device.[26] Training included hands-on device practice in the hospital's simulation center and a didactic program involving lectures on male anatomy, indications and contraindications for urinary catheter placement, and possible complications.[26] Criteria for proficiency included 5 practice procedures followed by 5 successful catheterizations, as determined by one of the physician investigators.[26] Among the 25 patients enrolled, there was a 100% success rate for Foley catheter placement, with minimal pain and 2 cases of gross hematuria.[26] On the basis of these results, a larger prospective, randomized study comparing the visually guided device to standard male urinary catheterization is being performed.[26]

The National Quality Forum listed direct visualization of the urethra during insertion of catheters as one of the safe practice innovations in their safe practice guidelines for catheter-associated urinary tract infection prevention developed in 2009, recognizing that damage to the urethra can occur with blind insertion, leading to the risk of infection.[4] Using direct visualization technology is in accordance with these established guidelines and benefits the patient and hospital staff. It has the potential to minimize patient pain, lower anxiety of both the patient and staff performing the catheterization, reduce iatrogenic injuries, avoid or reduce risks and costs associated with diagnostic imaging and advanced procedures, and reduce the need for urologist consultation.

Despite best efforts at problem solving, urologic consultations are necessary when the urethra cannot be entered owing to severe phimosis or meatal stenosis or if substantial resistance during catheter placement is encountered.[2] If kinking occurs in the urethra and a bloody discharge is present, urethral perforation may have occurred and a urologic consultation is required. In an attempt to minimize the extent of injury to the urethra and subsequent stricture formation that may require surgical reconstruction, prompt consultation for catheter placement appears appropriate in these circumstances. Numerous advanced techniques are available to the urologist for managing difficult catheterizations.[14] Blind glide wire techniques should not be performed by emergency physicians when interventional radiologists or urologists are available. Although this technique is well established in the literature, some urologists view it as controversial and consider flexible cystoscopy as the standard of care.[14] In a 2011 American Urologic Association update, Villaneuva and Hemstreet III[14] state that they only will perform such a technique after failure of 2 other techniques.

DISPOSITION

The stable, routine patient for whom catheterization was successful may be discharged home with urology follow-up after being fitted for a closed, leg-bag Foley system and educated about catheter management with home care. To prevent infection after catheter therapy, the integrity of the closed-catheter system should be maintained and the catheter

should be removed as soon as possible. Routine prophylactic antibiotics are not necessary, as use may promote resistance and complications.[2,15,27] However, 1 dose of oral antibiotic before discharge may be appropriate for certain patients,[2] such as those for whom excessive manipulation of the urinary catheter occurred. The duration of catheter placement is an area of debate but typically is 1 to 7 days, depending upon patient comorbidities (ie, diabetes mellitus, ambulatory impairment, prostatic enlargement, and expectation of resolution of initial need for catheterization).[24] One prospective study assessing the impact of catheter duration on voluntary voiding in men with AUR caused by BPH demonstrated that men with fluid retention volumes in excess of 1,300 mL had lower rates of failure with longer catheter duration.[28] On the basis of these findings, the authors recommended longer periods of catheter placement (7 days) for this group of patients to improve the likelihood of successful voiding.[28]

The complicated patient with systemic illness, such as fever, hypotension, or multiple comorbid medical conditions, will require hospitalization, as will patients with complications from manipulation or decompression.[16,29] An uncommon, yet frequently discussed condition, postobstructive diuresis, occurs in 0.5% to 52% of patients with chronic retention, but is not of clinical significance unless urine output of 200 mL/h occurs for more than 4 hours, which would necessitate hospitalization.[16,29] Limiting the amount of urine emptying and gradual drainage are not necessary, as hematuria occurs in 2% to 16% of patients after rapid, complete bladder emptying and most likely is of little clinical significance.[16,29] Hypotension occurs with bladder decompression, but again, does not seem clinically significant in otherwise healthy patients.[29]

Successful voiding after discharge also may be improved by pharmacologic therapy. Initiation of alpha blockers may provide sufficient decrease in smooth muscle tone at the bladder neck and in the prostate to allow successful voiding after catheter removal in men with significant prostatic enlargement, but should be used with instruction about the risk of side effects.[23,24,30–35] The most common side effects are dizziness and asthenia but also can include orthostatic hypotension, headache, nasal congestion, and delayed ejaculation.[23,24,30–35] Tamsulosin (0.4 mg orally daily) and alfuzosin (10 mg orally daily) are commonly used because they do not require titration dosing. Other options include phenoxybenzamine, prazosin, terazosin, and most recently, silodosin. Initiation of alpha blockers by the emergency physician is appropriate and will aid in the decision-making process of the urologist at the time of follow-up consultation regarding when to remove the urinary catheter if the patient passes a voiding trial. Patients with urinary retention after initiation of alpha blockers should undergo urologic consultation to assess therapy response and to exclude malignant disease as causative of urinary retention. Compared to placebo, long-term treatment with 5-alpha reductase inhibitors, such as dutasteride or finasteride, or a combination of finasteride and doxazosin, has been shown to produce a clinically significant reduction in total prostate volume and may prevent AUR in BPH.[23,24,33,34,36–38] The use of 5-alpha reductase inhibitors combined with an alpha blocker in a single oral dose now is available. The initial use of both agents is a decision that should be made after urologic consultation when it is clear that the patient will require prostate volume reduction, justifying the addition of another agent to treat voiding dysfunction.[23,34,35,37]

Complicated urinary catheterization is a commonly encountered medical problem, the frequency of which is difficult to estimate.[14] Patients have limited access to a clinician knowledgeable to treat this condition. Emergency physicians play a pivotal role by intervening in the acute presentation of genitourinary trauma, urinary retention, and the inability to decompress a urinary bladder. The tips and algorithm presented in this review offer guidance for improved success in blind catheter placement and also offer an advanced technique for high-risk patients by using direct visualization. Equipped with this insight, emergency medicine physicians and staff will be able to readily identify a problem with catheter placement and then have a solution available at the bedside to navigate successful catheter placement or conclude that a urologic consultation is needed. This approach offers patients better care, with less pain and complications, while minimizing hospital resources.

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Persistent Hiccups as a Rare Presenting Symptom of Pulmonary Embolism

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Pulmonary embolism (PE) is a life-threatening condition that may present as dyspnea, chest pain, cough or hemoptysis, but often occurs without symptoms. It is not typically associated with hiccups. Hiccups are generally self-limiting benign contractions of the diaphragm that may be associated with medications or food but may also be symptomatic of serious disease when persistent. We report 3 cases of PE presenting as persistent hiccups. [West J Emerg Med. 2012;13(6):479-483]

INTRODUCTION

Pulmonary embolism (PE) is a potentially lethal condition that can be difficult to diagnose. The incidence in the United States is as high as 1 per 1,000 persons per year, and it is the third commonest cause of death in hospitalized patients.[1] Early diagnosis and treatment are crucial in ensuring a good outcome, but symptoms are non-specific and the index of suspicion often low.[2-4] As a result the diagnosis is often made post-mortem.[5]

Hiccup or singultus is the involuntary, spasmodic contraction of the inspiratory muscles, especially the diaphragm. Hiccups are believed to be due to stimulation of the hiccup reflex arc and are generally transient and innocuous. Sometimes they are persistent and symptomatic of organic diseases in the nervous, respiratory, cardiovascular or digestive systems.[6-12]

The hiccup reflex arc consists of an afferent limb that includes the phrenic and vagus nerves, plus sympathetic fibers from T6-10; a center in the brainstem; and an efferent limb consisting mainly of the phrenic nerve. Irritation of any part of the arc in the head, neck, chest or abdomen can lead to hiccups. The irritant may be inflammation, medication, trauma or even over distension of a viscus.[6-8, 12, 13]

Hiccups have been associated with medications (steroids, dopamine, azithromycin, cefotetan, benzodiazepines, propofol),CNS disorders (tumors and vascular anomalies, multiple sclerosis and seizures), pulmonary disease (lung cancer), gastric and esophageal disease (GERD, herpetic esophagitis, gastric volvulus) and cardiac conditions (myocardial infarction, pacemaker lead injury).[7, 9, 12, 14-17] While many disease entities have been associated with hiccups, we found only one other report of pulmonary embolism presenting as hiccups.[28-33]

Pulmonary embolism may cause irritation of the afferent or efferent limb in the chest, although the exact mechanism that causes hiccups is unclear.

We report here 3 cases of pulmonary embolism presenting as persistent hiccups.

CASE DESCRIPTION

A search of the medical literature (search for PE and intractable hiccups through Medline, the Cochrane library database and Google search engine) did not reveal any case series of hiccups as a presenting symptom of PE, although a single citation describing this entity was identified in the lay literature and one case report.[33-34] In 2 of our 3 patients, recent surgery was a risk factor for PE.

Patient # 1

A 52-year-old African-American male without significant prior medical or surgical history presented to the emergency department (ED) with a 3-day history of hiccups. He had had progressively worsening dyspnea for 4 weeks, a dry cough and pain in the upper right back associated with inspiration and coughing. He had increasing leg edema for 2 weeks and was told he had congestive heart failure at another

institution, where he was advised admission, but left against medical advice. The rest of his review of systems (ROS) was unremarkable and he had no risk factors for deep venous thrombosis (DVT), such as family history of clotting disorder or personal history of immobilization, long journeys, surgery or trauma. He had quit occasional smoking many years ago and did not use alcohol or narcotic drugs.

Physical examination revealed a well-built male in mild respiratory distress with a respiratory rate (RR) of 20 breaths/minute, pulse rate (PR) of 108 beats/minute and blood pressure (BP) 113/76 mmHg. His oxygen saturation was 89-91% on room air. His electrocardiogram (EKG) was normal except for sinus tachycardia, and a duplex ultrasound of his lower extremities was negative for DVT. His portable chest radiograph (CXR) was the first hint of abnormality (Figure 1), showing a prominent pulmonary central artery (early Fleishner's sign, red arrow heads) and a pruned tree /cut off of the pulmonary arteries (Westermark's sign, black arrows). With the working diagnosis of PE, a computed tomography (CT) of the chest with intravenous (IV) contrast was performed which revealed a massive saddle embolus of the pulmonary artery (Figure 2A, black arrow). Patient was heparinized and transferred to a tertiary center where pulmonary thrombectomy was performed. He made a full recovery with resolution of hiccups and dyspnea. His laboratory examination with complete blood count (CBC), complete metabolic panel (CMP), prothrombin time (PT)/international normalize ration (INR) and partial thromboplastin time (PTT) was essentially normal. Workup for a coagulation disorder failed to reveal a reason for his embolus.

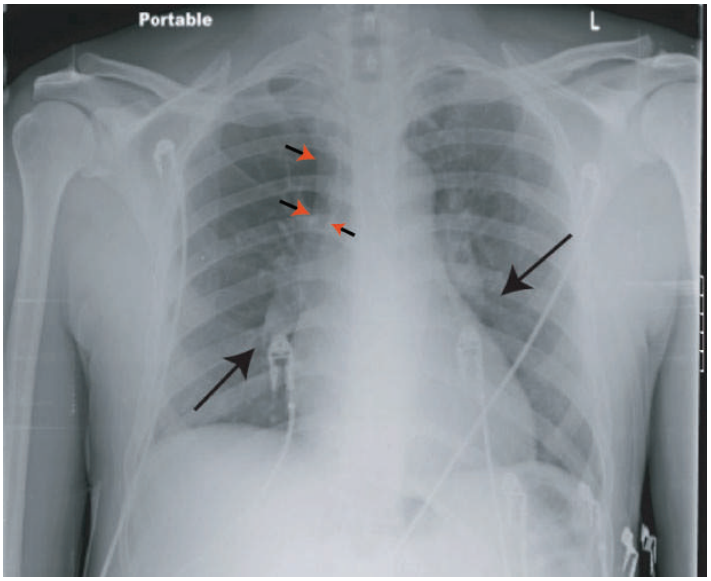

Figure 1. Chest radiograph demonstrating a prominent central pulmonary artery (early Fleishner's Sign, red arrows) and a cut-off of the pulmonary arteries bilaterally (Westermark sign, black arrows).

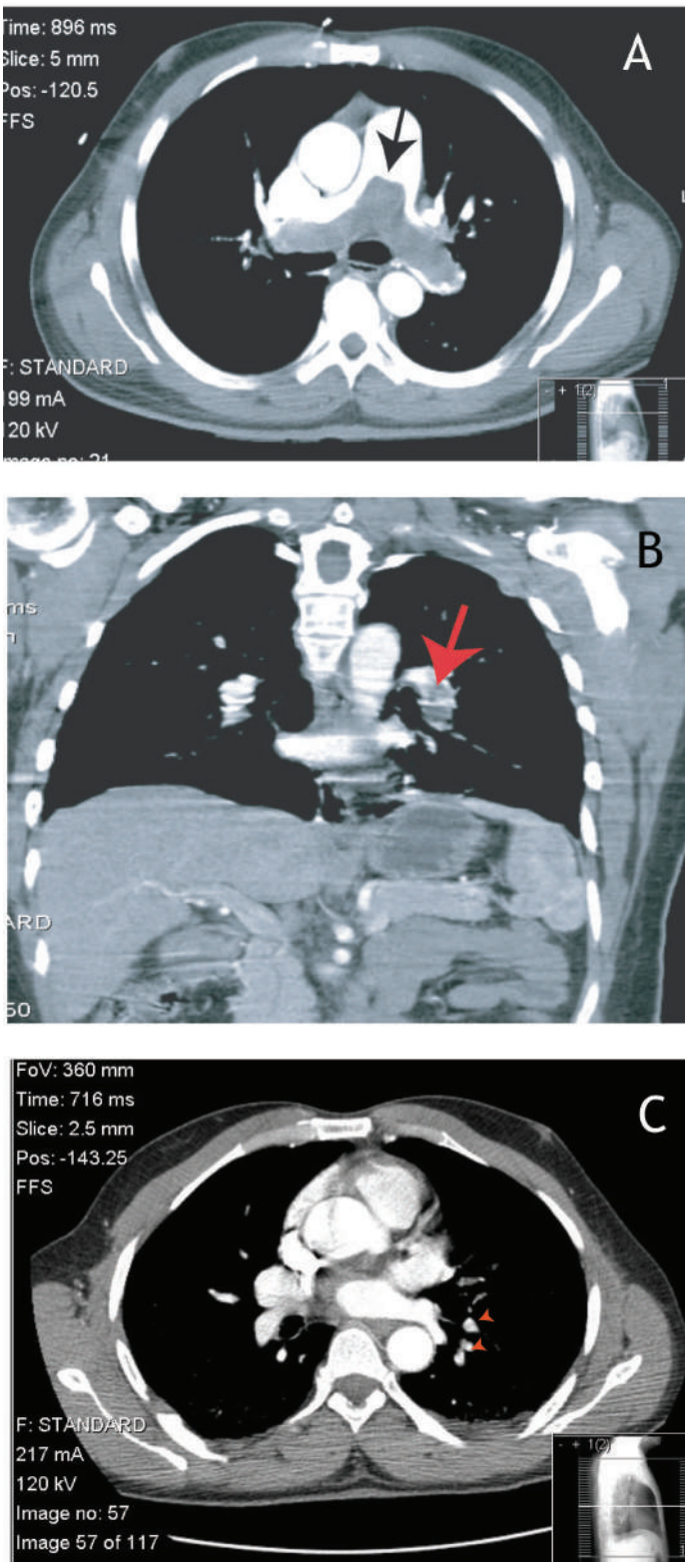

Figure 2. A. Computed tomography (CT) showing a saddle embolus (black arrow). B. CT showing a large left pulmonary artery embolus (filling defect, red arrow). C. CT showing a left pulmonary artery embolus (filling defect, red arrows).

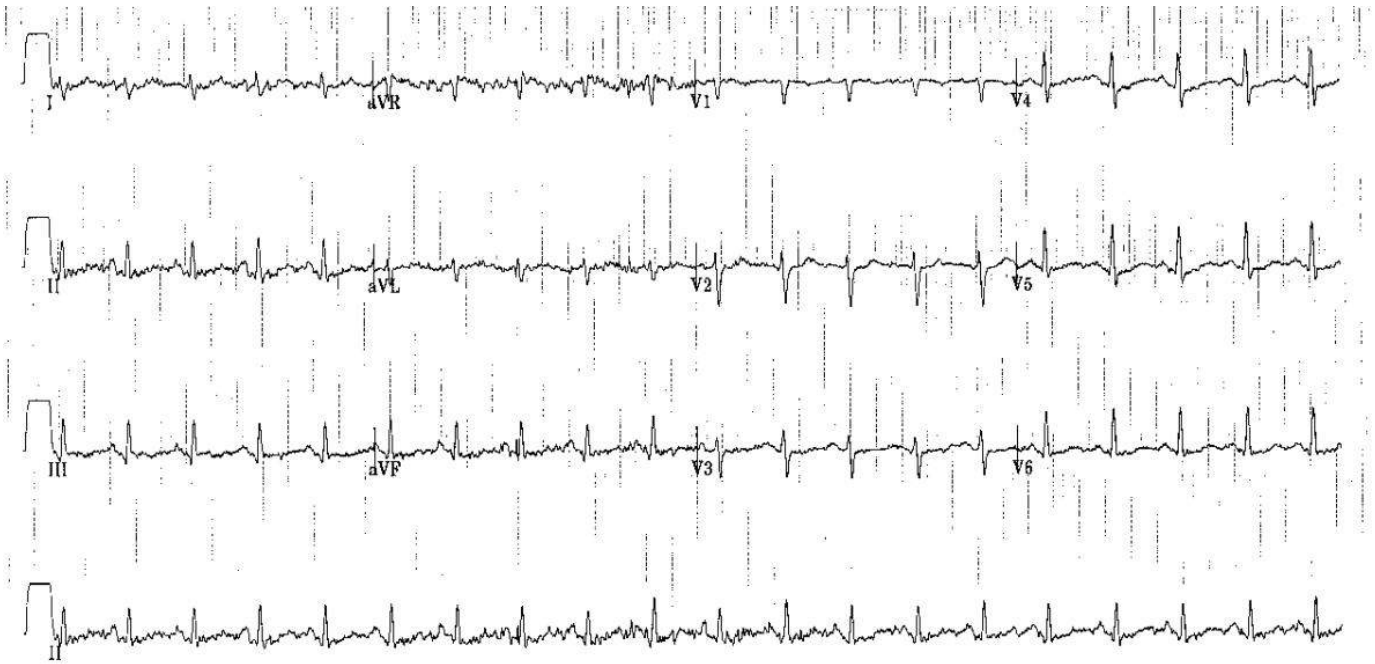

Figure 3. An electrocardiogram showing S1Q3T3 pattern indicating the likelihood for the presence of PE.

Patient # 2

A 40-year-old Hispanic female came for her follow-up gynecology appointment after undergoing total abdominal hysterectomy for fibroids under general anesthesia 12 days earlier. She complained of severe left lower back pain of 1-day duration and was referred to the ED to rule out renal colic. Her postoperative course was uneventful and she had been ambulating on post-operative day #1 and discharged home shortly after surgery. In the ED she spoke in fragmented sentences due to persistent hiccups. ROS and past medical history were unremarkable

Physical examination revealed a young female with hiccups and mild lower abdominal tenderness, but no signs of rhonchi, rales or crepitations in the chest. She was given dilaudid and morphine for back pain, but her PR was 110-120 beats/minute and BP 112/78 mmHg. Temperature was 100.7° Fahrenheit and RR 20 breaths/minute. Based on the previous case of PE presenting with hiccups, a diagnosis of pulmonary embolism was entertained. CXR was unremarkable, but EKG revealed S1T3Q3 changes (Figure 3). CT chest revealed a large left pulmonary embolus (Figure 2B, red arrow). Bedside duplex ultrasound did not show DVT. Her laboratory examination with CBC, CMP, PT, INR, and PTT was essentially normal. Workup for a coagulation disorder failed to reveal a reason for his embolus. She was started on IV heparin infusion and warfarin and discharged home with resolution of hiccups and back pain.

Patient #3

A 46-year old African-American male presented to the ED with persistent hiccups 2 days after having left inguinal herniorrhaphy under general anesthesia. Surgery was uncomplicated and performed as an ambulatory procedure. He had been ambulating at home, but one day after surgery developed hiccups. His past medical history was unremarkable, but he had undergone right inguinal herniorrhaphy in 2008 without any adverse events or symptoms. He was a nonsmoker and drank no alcohol and denied any other risk factors for DVT.

Physical examination revealed a middle-aged man in no apparent distress except for hiccups. At presentation his temperature was 98° Fahrenheit, PR 81 beats/minute, BP 128/77 mmHg and RR 18 breaths/minute. ECG showed normal sinus rhythm. He was given chlorpromazine, which resolved his hiccups, and he was discharged home. On his way home the hiccups returned and he came back to the ED. A second dose of chlorpromazine failed to resolve the hiccups this time. He also complained of feeling dizzy and his BP was found to be low. A chest CT was ordered and revealed a pulmonary embolus (Figure 2C, red arrow heads). His laboratory examination with CBC, CMP, PT, INR, and PTT was essentially normal. Workup for a coagulation disorder failed to reveal a reason for his embolus.

DISCUSSION

The symptoms and signs of PE are highly variable and non-specific and a high index of suspicion is critical in the diagnosis of this potentially lethal condition. The most common symptoms of PE are dyspnea at rest or on exertion (73%), pleuritic chest pain (44%), and cough (34%) with calf pain (44%) and calf or thigh swelling(41%) indicating DVT preceding PE.[35, 36] Patients with such symptoms or signs often need additional testing to confirm or exclude the diagnosis. Hiccups on the other hand are rarely associated with PE. Patients may also be completely asymptomatic, making the diagnosis more challenging. A meta-analysis of 28 studies found that among 5,233 patients who had DVT, 1,665 (32%) had asymptomatic PE, highlighting the importance of a high index of suspicion.[37] Risk stratification or pretest probability assessment thus becomes paramount in raising awareness of a possible diagnosis of PE.

All patients with chest pain or shortness of breath should have CXR and ECG. In patients with high pretest probability and non-specific symptoms, even if CXR and EKG are normal d-dimer level, lower extremity duplex scan, ventilation-perfusion (VQ) scans and CT angiogram may be needed.[35, 37-40]

Numerous prediction models and algorithms have been developed for assessment of patients with possible PE, including the Geneva, Kline, Pulmonary Embolism Rule-out Criteria (PERC), Pisa and Wells systems.[41-46] Of these the Wells criteria and the PERC rules are the most popular. The different elements included in these scoring systems are clinical history, physical examination, and diagnostic tests, such as arterial blood gas, D-dimer, ECG, and CXR with points ascribed to each element. Based on the total score and the clinical picture, the need for additional testing, such as VQ scans and CT angiograms, is determined by the clinician. Thus, by PERC rules if a patient meets all 8 criteria and falls into a low-risk category the probability of PE is < 2% and further testing may not be required. By Wells' criteria, a patient with low probability of PE and a negative D-dimer test may be discharged home without more testing.

Zylicz reported a case of intractable hiccups due to PE suggesting that a thrombus in the inferior vena cava (shown on ultrasound) caused PE and then hiccups, but no objective evidence of PE is presented.[33] Hiccups resolved with low molecular weight heparin, but recurred when heparin was discontinued. The patient also had underlying non-small cell lung cancer and it is unclear if lung cancer was the source of the hiccups. All of our patients had CT-documented PE and 2 had the risk factor of recent surgery.

Patient # 1 had a low Wells score of 1.5 (HR > 100), making him low risk, but he failed 3 of 8 PERC criteria and the CXR and overall gestalt raised suspicion for PE. Also the Kline decision rule (age/hypoxemia) put him in the unsafe category with a pretest probability of 45.2%. The revised Geneva score placed him in a moderate risk group based on tachycardia alone. Patient # 2 had a score of 3 on Wells criteria (HR > 100; recent surgery) and failed 2 of 8 PERC categories. In patient #3 Wells score was 1.5 (recent surgery), and he failed only 1 of 8 PERC categories.

Different scoring systems put patients in different risk categories highlighting the difficulty in relying only on one scoring system. Intractable hiccups in 2 patients raised our level of suspicion after our experience with patient #1, although both of these patients had recent surgery. CXR in patient #1 and EKG in patient # 2 also helped point us in the right direction. Our series of 3 patients with PE who had hiccups as a presenting symptom highlights the importance of including hiccups in the constellation of symptoms and signs associated with PE. If pretest probability testing is also taken into account, it will help us decide which patients need more advanced imaging and testing to confirm or exclude the diagnosis.

CONCLUSION

Hiccups are common and often idiopathic, but persistent hiccups should be taken seriously. They may be manifestations of immediately life-threatening conditions like myocardial infarction or even pulmonary embolism. Including hiccups in the pantheon of symptoms associated with PE will raise awareness as demonstrated by our small series.

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Takayasu's Arteritis - An Unusual Cause of Stroke in a Young Patient

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We describe the case of a 28-year-old-male with no significant medical history who presented with right-sided hemiparesis, bruits over the carotid and subclavian arteries and an elevated erythrocyte sedimentation rate. Imaging studies revealed a middle cerebral artery thrombus and inflammatory changes of the carotid and subclavian arteries and aorta. The diagnosis of Takayasu's arteritis was made and the patient was started on steroids and immunomodulators with good clinical response. [West J Emerg Med. 2012;13(6):484-487]

INTRODUCTION

While stroke is the third most common cause of death in the United States (US), affecting more than 2.4 million patients per year, only 3% of strokes occur in patients under the age of 40.[1-3] Atherosclerotic and embolic disease are common causes of ischemic stroke in both young and old patients. In the young however, a wider array of systemic and vascular diseases must be given consideration. Systemic inflammatory or autoimmune diseases, hypercoagulable states and vascular diseases such as dissection are responsible for about 20% of cases, while no certain cause is found in about one-third of young stroke victims.[3] Non-cerebrovascular entities must also be given consideration in the young patient presenting with symptoms suggestive of stroke. Todd's paralysis, complex migraines and conversion disorders are more likely to afflict the young and can manifest with a similar clinical picture.

CASE REPORT

A 28-year-old male with no significant past medical history presented to a community emergency department with acute onset of a dense right hemiplegia and aphasia. While at work the patient had a witnessed collapse to the ground. 911 was called.

A family member reported that the patient had been complaining of headaches and, back and shoulder pain for two weeks. No other systemic symptoms predated the patient's presentation. The patient had a history of mild intermittent asthma and used an albuterol metered dose inhalers as needed. There was no known family history of cardiac, cerebrovascular or autoimmune disease.

On presentation, the patient was acutely ill appearing. There were no visible signs of trauma. He had a blood pressure of 111/62 mmHg in the right upper extremity and 173/105 mmHg in the left upper extremity. His heart rate was 115 beats-per-minute and his respiratory rate was 14 breaths per minute with an oxygen saturation of 98% on 3L nasal cannula. He had loud bruits heard over the carotid and subclavian arteries as well as the abdominal aorta. There was a 3/6 systolic ejection murmur noted over the entire precordium. He was non-verbal, not following commands, and did not open his eyes spontaneously. He had extensor posturing to noxious stimuli on the right side.

The patient was intubated for airway protection and taken emergently for a non-contrast brain computed tomography (CT) that revealed a dense middle cerebral artery (MCA) sign on the left corresponding to a thrombus without acute hemorrhage, infarct or mass effect (Figure 1). The patient was administered alteplase within a 4.5 hour window without any immediate change in his clinical status.

A chest, neck and brain CT angiogram was performed, which showed marked wall thickening involving the left common carotid artery with complete occlusion of the distal left common carotid artery and MCA (Figure 2). Also seen was occlusion of the right common carotid and vertebral arteries with extensive collaterals, as well as inflammatory changes surrounding the aorta and subclavian vessels. No dissection of the aorta or its branches was visualized.

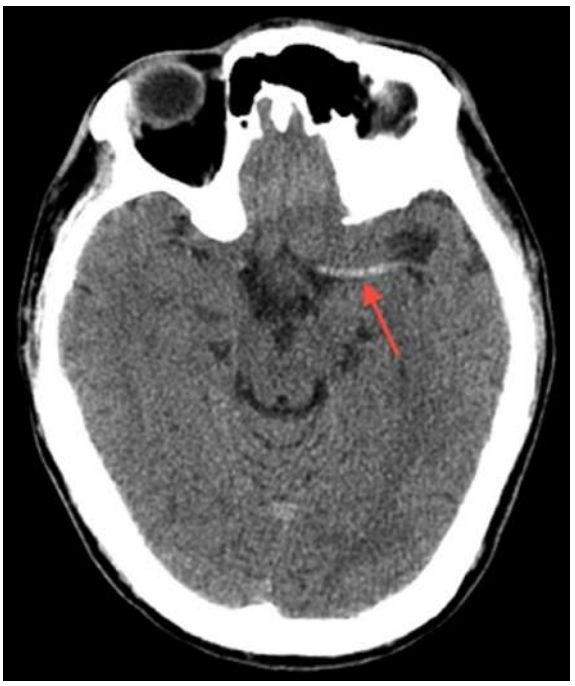

Figure 1. Non-contrast brain computed tomography showing a hyperdense lesion (arrow) corresponding to thrombosis of the left middle cerebral artery.

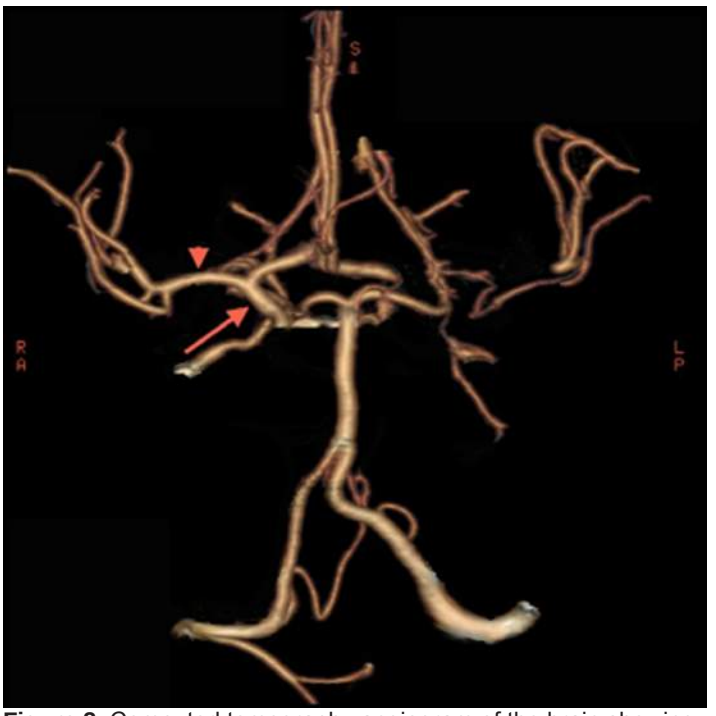

Figure 2. Computed tomography angiogram of the brain showing filling defects in the left internal carotid artery and left middle cerebral artery (MCA) corresponding to thrombosis of those vessels. A normally enhancing right internal carotid artery (arrow) and right MCA(arrowhead) are shown for comparison.

A brain magnetic resonance image (MRI) was performed demonstrating an evolving infarct involving the left deep basal ganglia, thalamus and cerebral cortex (Figure 3). There was restricted diffusion weighted signal of the left cerebral cortex signifying viable tissue with ongoing ischemia. As the patient had already received thrombolytic therapy and failed to improve, a neurosurgical consult was obtained and the patient was taken to the interventional suite where a thrombectomy, clot retrieval and angioplasty of the left common carotid and MCA thrombus was performed to allow for reperfusion (Figure 4).

Laboratory testing revealed an erythrocyte sedimentation rate (ESR) of 103 mm/hour (normal 0-15 mm/hr) and a C-reactive protein (CRP) of 51 (normal < 10). The patient's complete blood count, chemistries, coagulation profiles, hypercoagulable studies, electrocardiogram, and chest radiograph were normal.

Given the clinical exam, laboratory results and the findings on the CT angiogram, the presumptive diagnosis of Takayasu's arteritis was made. The patient was started on high dose steroids and methotrexate. He subsequently had normalization of his ESR and CRP and had marked clinical improvement by time of discharge.

DISCUSSION

Takayasu's arteritis, also known as pulseless disease, is a chronic inflammatory disease of unknown etiology that affects the aorta and its main branches. It was first described by Dr. Mikito Takayasu[4], a Japanese ophthalmologist, in 1905. In his report he described a 21-year-old woman with characteristic retinal arterio-venous anastomoses, syncope, and absent upper extremity pulses. Takayasu's arteritis is most commonly seen in females between the ages of 11 and 30 and is more common in Japan, Southeast Asia, India, and Mexico. It is rare in the US with an incidence of approximately 2.6 per million per year.[5]

The etiology of Takayasu's arteritis is unknown, but evidence suggests an autoimmune process, given the association with certain human leukocyte antigen (HLA) alleles and other autoimmune processes such as sarcoidosis and inflammatory bowel disease. It is also suggested that tuberculosis may have an association, given a high prevalence of active and past infection in patients with Takayasu's arteritis.[6]

Takayasu's arteritis typically presents in a pre-pulseless phase with non-specific systemic inflammatory features, such as fever, night sweats, malaise, weight loss, and arthralgias, followed by a chronic, pulseless phase with the development of vascular insufficiency and compromise. Morbidity and mortality are mainly the result of vascular occlusion and end organ damage. In the pulseless stage the typical manifestations of disease depend on the vessels involved. The most common manifestations of Takayasu's arteritis are limb claudication and ischemia from peripheral vascular involvement, hypertension from renal artery stenosis,

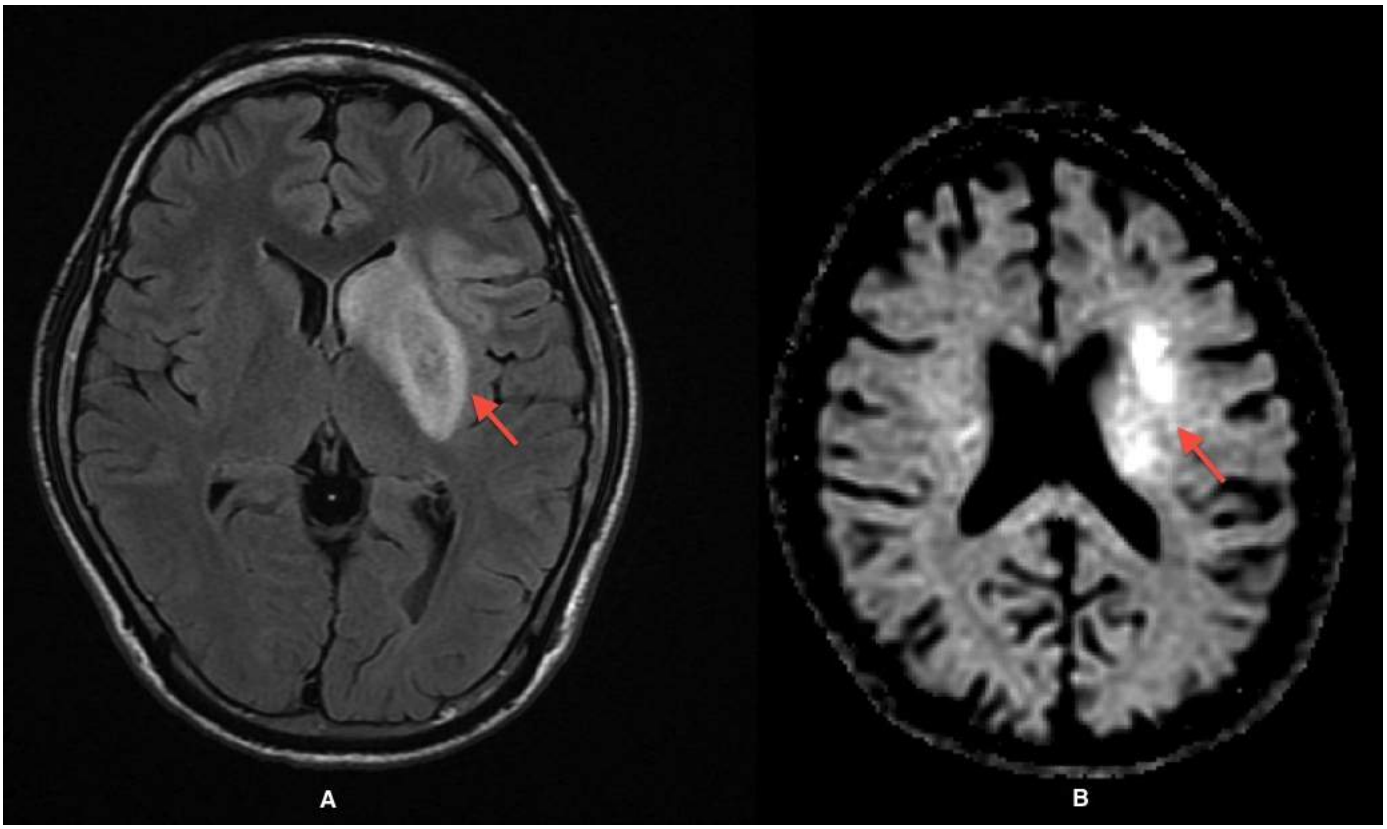

Figure 3. (A) T1-weighted and (B) diffusion weighted magnetic resonance image of the brain showing an evolving infarct (arrows) involving the left-sided deep basal ganglia, thalamus and cerebral cortex.

ophthalmologic disease as manifested by retinopathy or amaurosis fugax, aortic regurgitation resulting from dilatation of the ascending aorta, cardiac ischemia or congestive heart failure as a result of hypertensive and aortic disease,

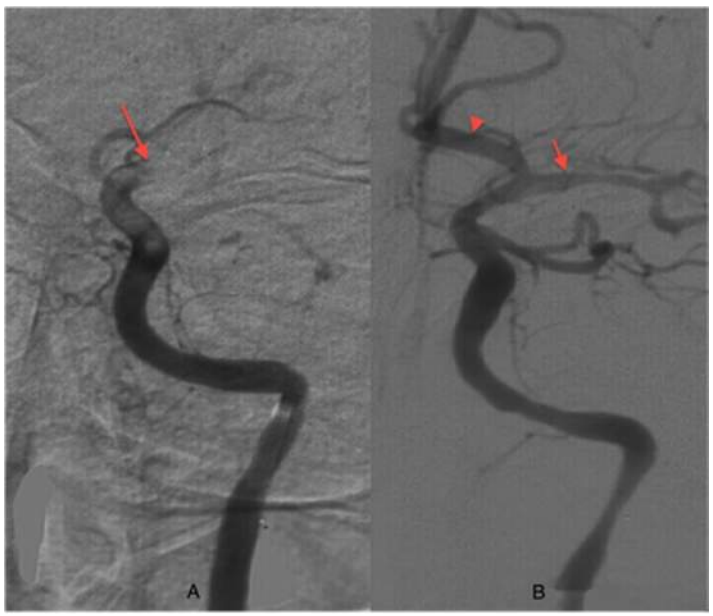

Figure 4. (A) Conventional angiogram showing occlusion of the left common carotid artery (long arrow) and (B) after thrombectomy with reperfusion of the middle cerebral artery (short arrow) and anterior cerebral artery (arrowhead).

pulmonary hypertension from pulmonary arterial involvement, and lastly neurologic disease (seizures and stroke) as a result of intra and extra-cranial arterial inflammation or thrombosis. Strokes are a common complication of Takayasu's arteritis with an estimated incidence of 10-20%.[7] Stroke as the first manifestation, however, is rare and few case reports exist in the literature.

In 1990, the American College of Rheumatology proposed criteria for the diagnosis of Takayasu's arteritis: age at disease onset ≤ 40 years, claudication of the extremities, decreased brachial artery pulse, blood pressure difference >10 mmHg in the upper extremities, bruit over subclavian arteries or aorta and arteriogram abnormalities. The presence of three out of six criteria are required for diagnosis and demonstrate a sensitivity of 90.5% and a specificity of 97.8%.[8]

No known serologic marker exists to diagnose or track progression of disease. ESR, the most commonly used marker to identify systemic inflammatory conditions, is not a reliable indicator and has been found to underestimate disease activity when compared to arterial histopathology or angiography.[7] CT, doppler angiography and MRI/magnetic resonance angiography can aid in diagnosis and can be used to track disease progression and response to therapy.

Immediate priorities in treatment must address acute vascular compromise through either medical treatment with thrombolytics or surgical or interventional

revascularization. Long term treatment is targeted at decreasing vessel inflammation and progression of vascular disease and controlling comorbid conditions. Glucocorticoids are the mainstay of anti-inflammatory treatment. Patients not responding to steroid therapy can be treated with immunomodulators, including methotrexate, cyclophosphamide and azathioprine. Despite long term treatment with corticosteroids and cytotoxic drugs, many patients will still experience progressive vascular disease. Medical management does not generally reverse pre-existing vascular stenosis or occlusion and therefore revascularization may be necessary if hemodynamically significant lesions occur.[8,9] Five-year survival is estimated to be 80% and largely depends on the clinical manifestations of disease and response to medical and surgical therapy.[10]

Our patient had only non-specific arthralgias and headache prior to his stroke. At the time of diagnosis, he had significant disease as demonstrated by the angiographic findings on CT and angiography. He had five out of six of the criteria set forth by the American College of Rheumatology and was thus diagnosed with Takayasu's arteritis. Treatment was started accordingly with high dose steroids and methotrexate. On discharge from the hospital, the patient had regained a significant degree of function, with 4 out of 5 motor strength on the right side and a mild residual expressive aphasia. He was discharged to an inpatient rehabilitation facility for aggressive physical, speech and occupational rehabilitation. Six months after discharge, on routine follow-up, the patient continued to have significant differential blood pressures in his upper extremities as well as bruits over his subclavian and carotid vessels. A follow-up CT angiogram of the brain and neck was performed and, despite normalization of the ESR and CRP, showed continued significant wall thickening of the subclavian and carotid arteries and aorta as well as severe re-stenosis of the left common carotid artery. The patient underwent successful stenting of the lesion. He was continued on methotrexate 25 mg weekly and prednisone 7.5 mg daily with the plan for interval CT angiograms to track the progression of vascular disease.

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Unusual Presentation of *Toxoplasma Gondii* Encephalitis

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We report a case of altered mental status secondary to acute *Toxoplasma Gondii* encephalitis. The patient had no medical or surgical history and presented with acute onset of lethargy with no clear precipitant. A physical exam revealed no focal neurological deficits and a subsequent medical workup revealed multiple intracranial lesions with a biopsy confirming the diagnosis of *Toxoplasma Gondii* encephalitis in the setting of newly diagnosed human immunodeficiency virus (HIV). A literature review revealed that this is a unique case of toxoplasmic encephalopathy in the United States in a previously undiagnosed HIV positive patient presenting to an emergency department. [West J Emerg Med. 2012;13(6):488-490]

A 33-year-old Yemenese male presented to the emergency department (ED) accompanied by family members who relayed a 2-day history of fever, headaches, nausea, vomiting, and confusion. The patient had increased sleepiness and progressive mental status changes over the preceding 48 hours, and the family had noted that he had become more disoriented and lethargic in nature.

There was no reported medical or surgical history, although the patient was recently treated as an outpatient for community-acquired pneumonia. The patient and present family members denied any history of depression, drug use, sick contacts, or trauma. The patient had resided in the United States for the past 10 years and had not traveled outside of the country in that time.

On physical examination the patient was afebrile with normal vitals. The patient would respond "yes" or "no" to questions, but would not speak in complete sentences. The Glasgow Coma Scale (GCS) was 13, with points subtracted for opening his eyes to only pain. Physical exam revealed white plaques in the intra-oral cavity. A focused neurologic exam, to the best of the patient's ability to cooperate, revealed no focal deficits. The patient's cranial nerves were intact and he was moving all extremities with no noted weakness or decrease in sensation. His reflexes were normal and there was no ataxia present. Other systems, including cardiac, respiratory, abdominal, and genitourinary examination, were normal.

A differential diagnosis for the patient's presentation included concern for drug or alcohol intoxication, endocrine abnormalities, central nervous system (CNS) infection, toxic ingestion, sepsis, ischemic or hemorrhagic stroke, uremia, and CNS tumors.

Laboratory tests revealed normal blood glucose, negative urine drug screen, normal electrolytes, and normal complete blood count. Additionally, a blood alcohol was negative, as well as a urine toxicology screen, and the patient had a normal anion gap and osmolar gap. A head computerized tomography (CT) was obtained (Figure 1). The CT showed multiple areas of vasogenic edema involving the supratentorial brain parenchyma with the largest of these involving the left anterior/inferior frontal lobe, right parietal lobe, left thalamus, right frontal and temporal lobe and right perisylvian region. There was also noted mass effect on the left frontal horn and right temporal horn and for this reason a lumbar puncture was not performed in the ED. Urgent magnetic resonance imaging of the head was obtained with gadolinium, which demonstrated multiple solid and irregular rim enhancing lesions with surrounding vasogenic edema (Figure 2).

The patient was taken by neurosurgery service to the operating room where a biopsy was taken, which was positive for *Toxoplasmosis gondii*. The patient was then found to be positive for the human immunodeficiency virus (HIV) with subsequent testing. While in the intensive care unit the patient required mannitol, steroids, and hypertonic saline for cerebral edema, and his neurologic status improved with treatment. The patient was treated for cerebral toxoplasmosis, subsequent *Pneumocystis carini* pneumonia, oral thrush, and was started on highly active anti-retroviral therapy (HAART).

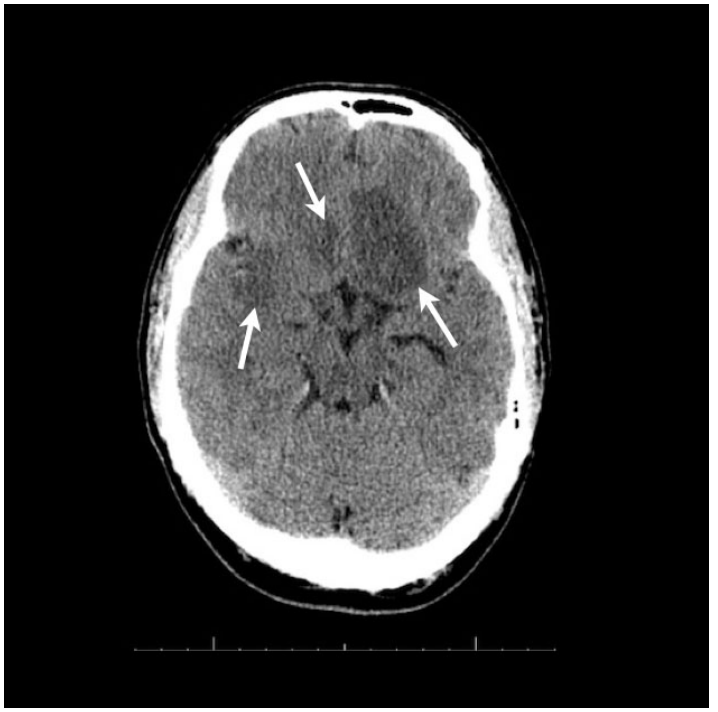

Figure 1. Head computed tomography of patient demonstrating areas of intracerebral vasogenic edema (arrows).

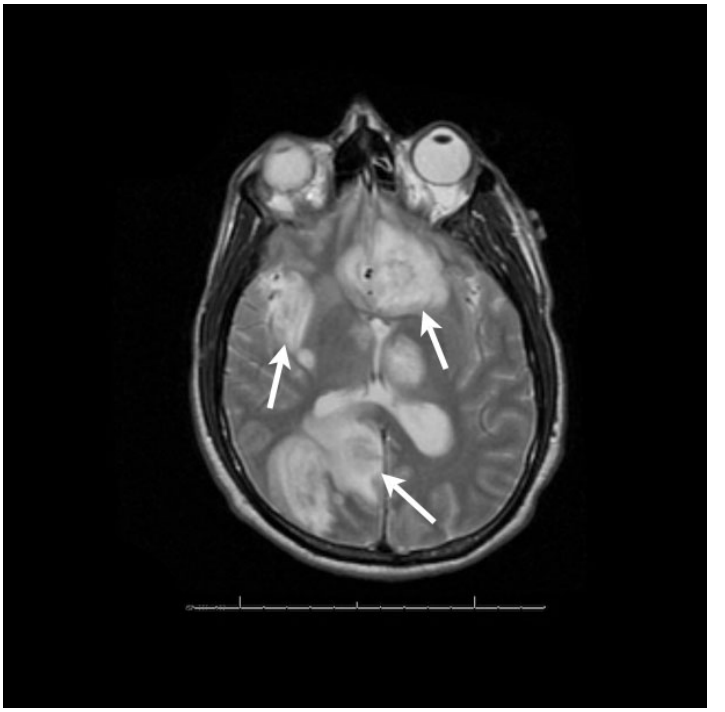

Figure 2. Magnetic resonance imaging with gadolinium with multiple enhancing lesions (arrows).

Toxoplasmosis gondii is an opportunistic intracellular pathogen that has long been recognized as the most frequent cause of brain lesions in acquired immune deficiency syndrome (AIDS) patients.[1,2] Seroprevalence of *Toxoplasmosis gondii* is estimated at 22.5% within the United States (U.S.) and may be higher within the general populous in Europe and tropical countries.[3,4] Infection with *Toxoplasmosis gondii* in adults with normal immune function will normally be asymptomatic or cause mild complaints of fever or malaise, while in the immunocompromised host it can cause severe central nervous system infections. Up to 10% of newly diagnosed AIDS patients can present with neurologic toxoplasmosis with the classic finding on head CT of a ring-enhancing lesion.[5] In the days prior to HAART treatment and rapid human HIV testing, up to 10-20% of newly diagnosed HIV patients presented with neurologic disease.[6]

A review of the literature shows that this is a unique case of toxoplasmic encephalopathy in the U.S. in a previously undiagnosed HIV positive patient presenting to an ED. Garcia-Gubern et al[7] noted a case of toxoplasmic encephalopathy of the spinal cord; however, it was different than our case because the patient had focal neurologic deficits of the lower extremities. Lee et al[8] relate a case of cerebral and optic nerve toxoplasmosis in an undiagnosed HIV positive patient, but this case differs in that the patient had neurologic symptoms manifested by vision loss. In a case reported by Snyder[9], a 27-year-old male was diagnosed with CNS toxoplasmosis, although the patient was exhibiting right-sided weakness and had known risk factors associated with the acquisition of HIV. Jayawardena et al[10] reported a case of a 35-year-old female with cerebral toxoplasmosis, but the patient was already known to be HIV positive. Therefore, this is the first published case report of toxoplasmic encephalopathy in a previously undiagnosed HIV positive patient presenting to an ED with altered mental status without focal neurological deficits and no known risk factors associated with the acquisition of HIV.

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Bilateral Thalamic Infarction

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A 35-year-old woman, without previous medical history except oral contraception, presented with sudden onset of stupor and clonic perseveration in the upper limbs. She was aphasic, but communicated by vertical movements of the head. Unenhanced brain computed tomography (CT) demonstrated hyperintensity of the straight sinus and hypo-intense areas within both thalami (Figure 1A) leading to a diagnosis of cerebral venous thrombosis (CVT). Despite anticoagulation therapy, the patient deteriorated with a Glasgow Coma Score at 7. A brain magnetic resonance imaging (MRI) obtained two days later confirmed extensive acute venous thrombosis involving the straight sinus, the right tentorial sinus, the left proximal tentorial sinus and the vein of Galan. A bilateral thalamic venous infarction was confirmed (Figure 1B). Local thrombectomy followed by thrombolysis within the straight sinus was unsuccessful. A mild hemorrhagic transformation of the bithalamic infarction was observed on follow-up MRI. Neurological status slowly improved over time. At 3-month follow-up, she had no cognitive deficit and was able to walk in spite of residual spasticity.

Bilateral infarctions of the thalami may be observed from either arterial or venous origin. They account for only 0.6% of all cerebral infarctions. Occlusion of the top of the basilar artery or of the so-called artery of Percheron – a developmental variant replacing the perforating medial thalamic arteries – is responsible for arterial ischemia. On venous side, the posterior group of thalamic veins drains into the straight sinus. A thrombosis of this vessel may also lead to a bilateral partial venous infarct due to the upstream overpressure.

The diagnosis of CVT is often challenging, as clinical symptoms are highly variable and unspecific.[1] Good clinical recovery may be observed, even after severe and prolonged deficits at early acute stage of the disease course. Common risk factors for CVT, when identified, include pregnancy, early post-partum, oral contraception, hypercoagulability and infection.

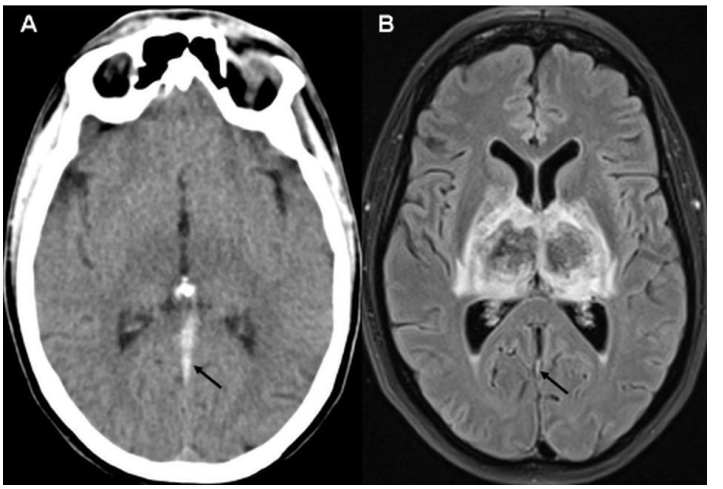

Figure 1. A. Unenhanced brain computed tomography. Strong hyperintensity within straight sinus due to the presence of an acute clot (arrow) together with subtle hypo-intensity within both thalamomesencephalic junctions. **B.** Magnetic resonance imaging with Fluid Attenuated Inversion Recovery (FLAIR) sequence. Hemorrhage within both thalami (central hyposignal intensity due to the presence of deoxyhemoglobin and marginal hyper signal intensity due to surrounding vasogenic edema). Thin residual clot within straight sinus (arrow) and moderate enlargement of lateral ventricles.

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Definitely NOT Just another Hernia

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A 77-year-old male presented to our emergency department (ED) with a 3-hour history of acute severe lower abdominal pain, inability to void, a swollen scrotum, and one episode of vomiting. His relevant past medical history included: hypertension, diabetes mellitus, and distant right inguinal hernia repair. Vital signs were: 96.4^0 F, 101, 22, and 205/90. Examination revealed a normal abdomen and an irreducible left inguinal hernia. Foley catheter placement yielded a scant amount of infected urine that stopped flowing almost immediately and did not resume despite catheter flushing and repositioning. Additional laboratory data did not identify the cause of the patient's symptoms. An abdominopelvic computed tomography (CT) was performed (Figures 1 and 2) after which the patient underwent emergent operative reduction of his incarcerated scrotal cystocele.

Complete bladder herniation into an inguinal hernia sac is referred to as a scrotal cystocele. This very rare condition is previously unreported in the emergency medicine literature. Some bladder involvement occurs in 1 - 4% of all inguinal hernias[1] and is often an incidental (generally benign) operative finding. Patients with partial bladder herniation may report "double phase micturition." This occurs when the patient voids then passes more urine by applying pressure to their hernia.[2]

Scrotal cystocele can be associated with bilateral hydronephrosis, acute renal failure, bladder lithiasis and necrosis, vesicoureteral reflux, and scrotal abscess.[1] This inherently serious condition mimics the far more common inguinal hernia containing fat or bowel. Risk factors include: patients over 50 years of age, males, and obesity. ED ultrasound may strongly suggest or confirm the diagnosis. CT is definitive.

Due to its associated morbidity, emergency physicians should consider scrotal cystocele in males with obstructive uropathy and an associated inguinal hernia.

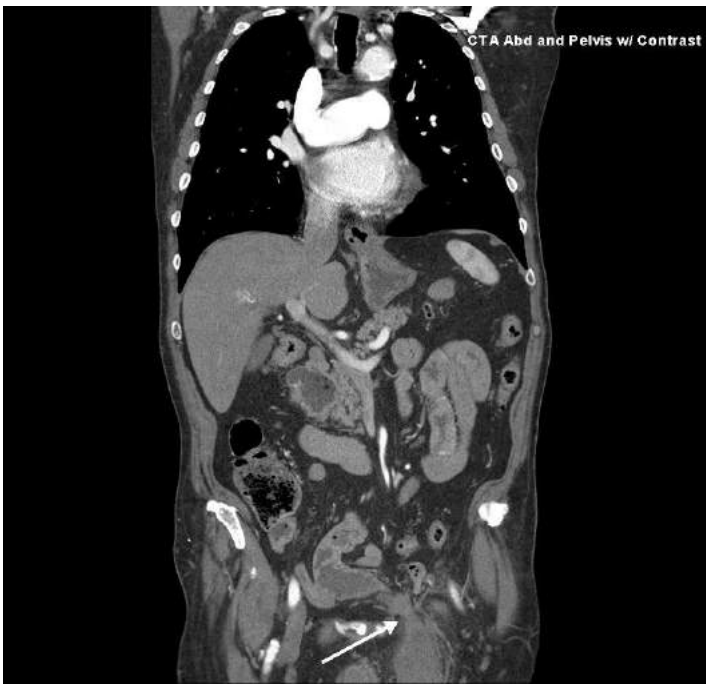

Figure 1. Scrotal cystocele-transverse view. Bladder entering inguinal canal. Arrow marks focal volvulus.

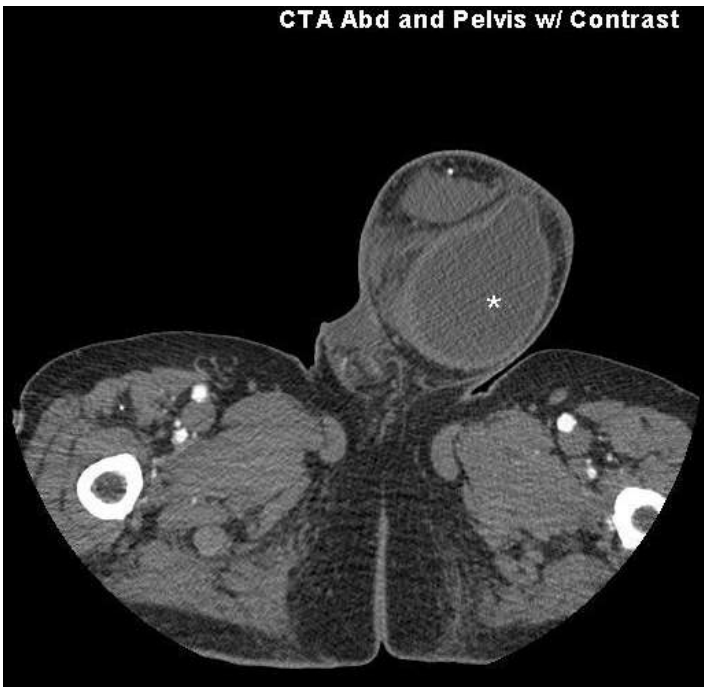

Figure 2. Asterisk marks distal obstructed bladder.

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Chief Complaint: Right Hip Pain

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A 50-year-old woman with a history of non-insulin dependent diabetes mellitus (NIDDM) presented to the emergency department (ED) with right hip pain for 1 week. The pain was described as constant, non-radiating, and worse with weight bearing. She denied any trauma, fevers, intravenous drug use, or recent surgery. Physical exam revealed a well-appearing afebrile patient with an antalgic gait slightly favoring her left side. The patient did not have hip or back tenderness, pain with axial loading, or ranging of the hip or knee. Labs were notable for glucose 465, WBC 19.1 with 12% bands. Computed tomography (CT) with intravenous contrast of abdomen and pelvis was done to assess for a possible psoas abscess.

SPINAL EPIDURAL ABSCESS

First described in 1761, spinal epidural abscess is a rare diagnosis occurring in 0.2-2 cases per 10,000 hospital admissions[1]. The classic triad of back pain, fever, and neurologic symptoms is specific however not sensitive, 99% and 13% respectively, as found in one study.[2] The imaging modality of choice is magnetic resonance imaging (MRI) however, it is occasionally visualized on CT.[3] Risk factors include an immunologically compromised host, concomitant distant site of infection, or recent back trauma or instrumentation.[2] Treatment consists of broad-spectrum IV antibiotics and neurosurgical decompression.[4]

CASE CONCLUSION

The CT revealed gas within the paraspinous musculature and epidural space of the lumbar spine. Along with an epidural abscess with gas-forming bacteria, the differential diagnosis includes discitis and degenerative spine disease. Broad-spectrum antibiotics, confirmatory MRI and neurosurgical consultation were initiated immediately and the patient did well with treatment.

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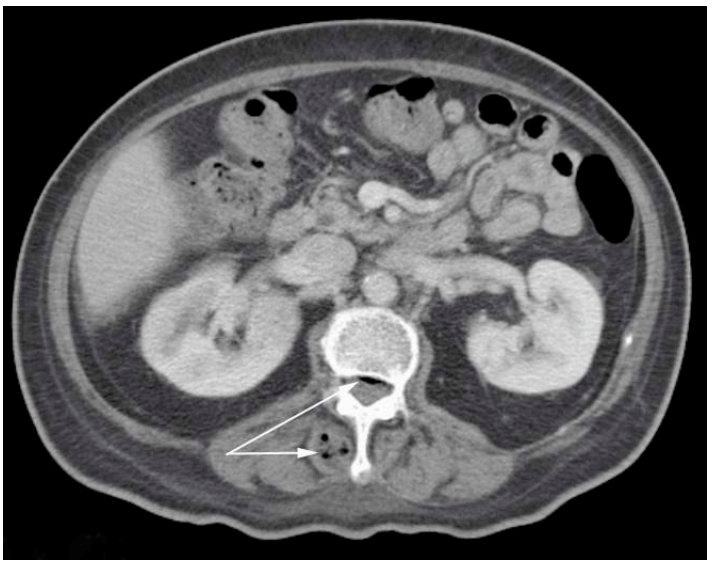

Figure. The air in the right paraspinous musculature and spinal column seen on the computed tomography are the first evidence of the lumbar spinal epidural abscess.

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Left Upper Quadrant Abdominal Pain

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We present a case of acute appendicitis from mobile cecum presenting with left upper quadrant abdominal pain. [West J Emerg Med. 2012;13(6):495-496]

A 58-year-old male with history of hypertension presented with constant left upper quadrant (LUQ) pain for 3 days. He denied fevers, chills, had no anorexia or association with eating, and no changes in bowel or bladder function. He was nontoxic appearing with normal vital signs. Abdomen was soft with tenderness to palpation in LUQ with normal bowel sounds. There were no masses, guarding, or other areas of tenderness. Remainder of the examination was normal. White blood count was 10,600/mm^3 with 74.9% neutrophils with otherwise normal indices. Serum chemistry, urinalysis, and liver function tests, including lipase, were all in normal range. C-reactive protein (CRP) was 217 mg/L (reference range: 0-7 mg/L). A computerized tomography (CT) with intravenous contrast was obtained.

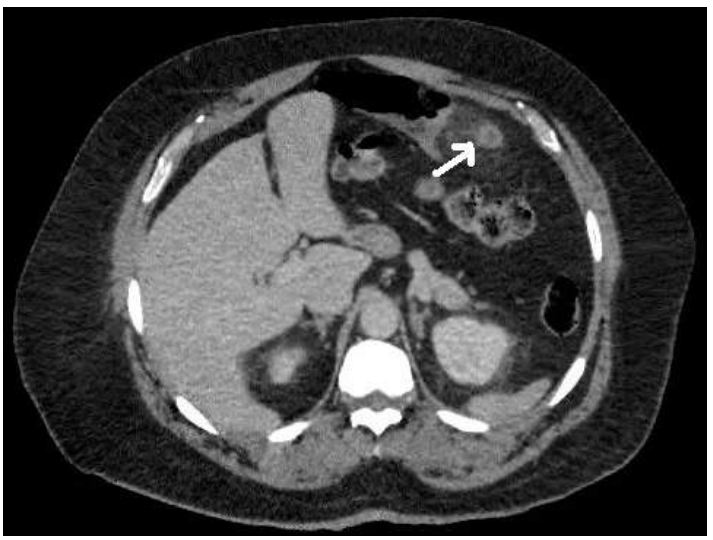

Figure 1. Axial image identifying left-sided enlarged appendix (arrow).

Diagnosis: Left-sided Appendicitis

The CT demonstrated a mobilized mesocecum in the left upper quadrant with a large, inflamed thick-walled appendix consistent with acute appendicitis. He underwent a laparotomy. The colon and cecum were found in the left upper quadrant with an inflamed, enlarged appendix. No malrotation or other congenital abnormalities were seen. Histology demonstrated a gangrenous appendix. He tolerated surgery well and was discharged home two days later.

Left-sided acute appendicitis is predominantly seen with

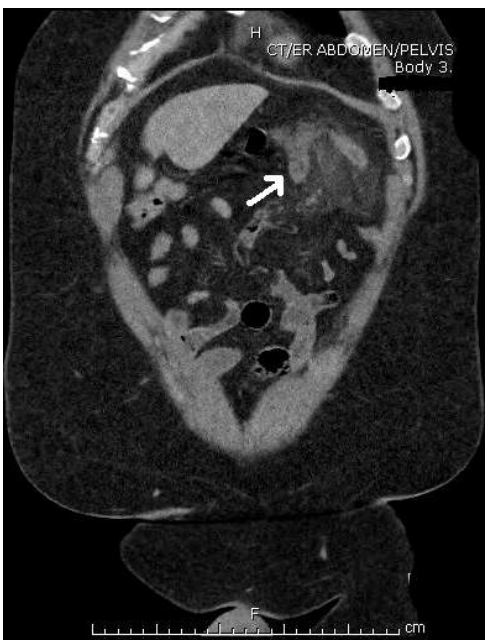

Figure 2. Coronal image identifying left upper quadrant acute appendicitis (arrow).

congenital anomalies, e.g., situs inversus totalis and midgut malrotation.[1] The majority of these present with left lower quadrant pain.[1] The patient presented had LUQ pain due to a hypermobile colon. Though not specific, an elevated CRP has been suggested as a marker of acute appendicitis.[2] The elevated CRP and clinical concern led to obtaining the CT.

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What's Eating Your Wishbone? Sternoclavicular Septic Arthritis with Osteomyelitis

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A 63-year-old female with a past medical history of hepatitis C and cirrhosis presented to the emergency department with a four-month history of increased swelling and tenderness to the right clavicle. The patient was afebrile with a firm, warm area measuring 6 cm x 4.5 cm x 2 cm, located over the medial right clavicle. The patient had a remote history of minor trauma that resulted in no fractures and required no interventions. She was diagnosed with sternoclavicular (SC) septic arthritis, with underlying osteomyelitis of the clavicle and sternum noted on plain radiographs and confirmed by computed tomography (CT).

Septic arthritis of the SC joint accounts for only 1% of all septic arthritis in the general population, but up to 17% in intravenous (IV) drug users. Common risk factors include IV drug use, distant site of infection, diabetes mellitus, and thoracic central venous line (CVL) placement. Less common predisposing conditions include minor trauma, cellulitis, cirrhosis, and immunocompromised states. Unlike the acute presentation of other septic joints, SC septic arthritis usually develops more slowly over 14 days.[1,2]

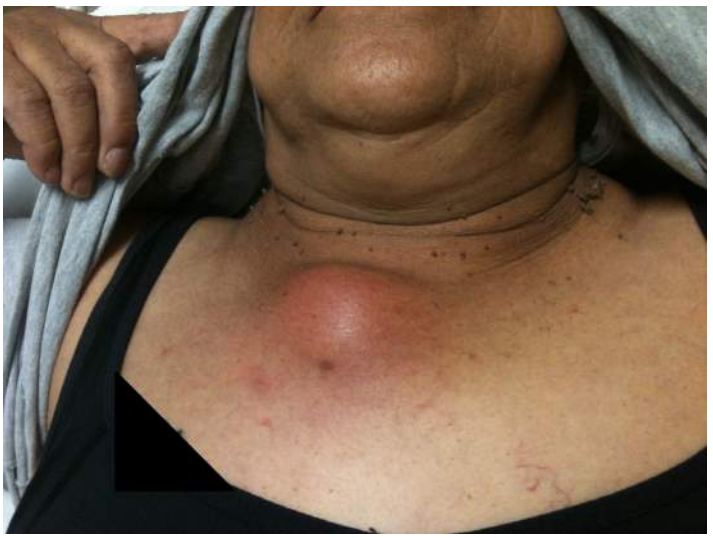

Figure 1. Photograph of patient's wound when she presented to the emergency department.

Patients suspected of having SC joint septic arthritis require either CT (83% sensitive) or magnetic resonance imaging (nearly 100% sensitive) to identify SC joint infections.[2] Blood and wound cultures guide antibiotic treatments; wound cultures may be obtained by simple incision and drainage, but usually require CT-guided needle aspiration.[3,4]

Patients with radiologic evidence of SC joint infection should be treated aggressively with IV anti-staphylococcal antibiotics, based on local sensitivities. Gram-negative coverage should be added in IV drug users, immunocompromised, and patients with CVL infections.[3] Antibiotic coverage should then be tailored based on blood and aspiration cultures of the affected SC joint. The majority of SC joint infections will require surgical debridement.[4]

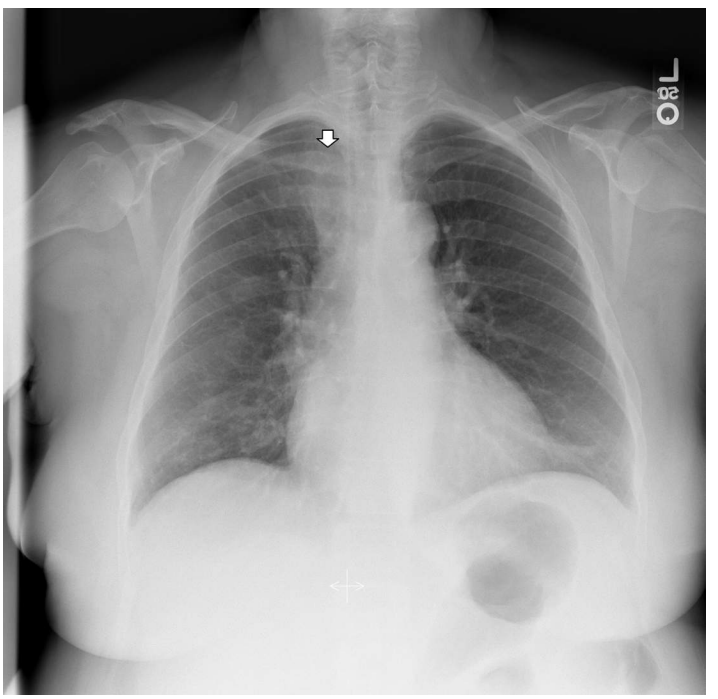

Figure 2. Chest radiograph of patient showing abnormality at the right sternoclavicular joint (arrow).

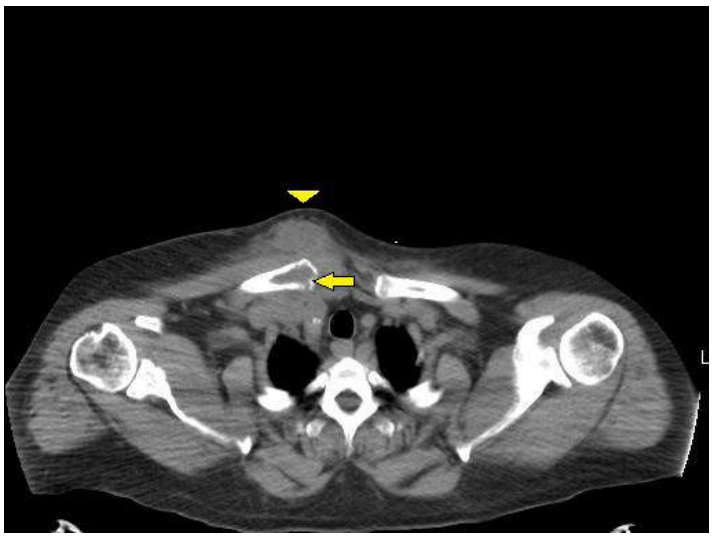

Figure 3. Non-contrast computed tomography of chest showing erosion of the right medial clavicle (arrow) with overlying soft tissue swelling (arrow head).

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Purple Urine Bag Syndrome

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A 67-year-old woman presented to the emergency department in congestive heart failure. She also had a history of transverse myelitis, which had caused her to be bedbound with an indwelling urinary catheter. During the physical examination, the urine in her Foley tubing and bag were noted to be bright purple. When asked, she replied that the urine had turned purple several weeks earlier. She denied suprapubic pain, but did complain of terrible constipation. Her urinalysis was significant only for a pH of 9, many bacteria, and 4 RBC/hpf. Her urine culture later grew >100,000 organisms/mL *Morganella morganii* and >100,000 organisms/mL *Proteus mirabilis*.

Purple Urine Bag Syndrome (PUBS) is an uncommon syndrome that occurs predominately in constipated bedbound women with chronic indwelling Foley catheters in which both the urine and catheter set become purple.[1] The cause of this color change is still not completely known, but is thought to arise from a complex series of chemical interactions resulting from concurrent constipation and urinary tract infection (UTI).[2,3]

Constipation allows adequate time for gut flora to deaminate dietary tryptophan to indole. Indole then travels via the portal system to the liver, where it is conjugated to indoxyl sulfate. This is subsequently excreted into the urine, where bacterial indoxyl sulfatases catalyze it to indoxyl. This metabolite then oxidizes to both indigo (blue) and indirubin (red) in the presence of a high urinary pH.[1,2] These pigments interact with the plastic of the catheter set to create a purple hue.[4]

Common causative bacteria are *Proteus mirabilis, Pseudomonas aeruginosa, Klebsiella pneumoniae, Escherichia coli,* Morganella, and Enterobacter spp.[1,4] While overall a benign condition, PUBS should signal to the clinician the presence of an underlying UTI that should be treated to prevent serious complications.[1,5] This includes antibiotic administration and improving Foley catheter care and hygiene.[1,2]

Figure. Purple urine and catheter set.

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An Uncommon Case of Abdominal Pain: Superior Mesenteric Artery Syndrome

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Superior mesenteric artery (SMA) syndrome is a rare cause of abdominal pain, nausea and vomiting that may be undiagnosed in patients presenting to the emergency department (ED). We report a 54-year-old male presenting to a community ED with abdominal pain and the subsequent radiographic findings.The patient's computed tomgraphy (CT) of the abdomen and pelvis demonstrates many of the hallmark findings consistent with SMA syndrome, including; compression of the duodenum between the abdominal aorta and superior mesenteric artery resulting in intestinal obstruction, dilation of the left renal vein, and gastric distension. Patients diagnosed with SMA syndrome have a characteristically short distance between the superior mesenteric artery and the aorta (usually 2–8 mm) in contrast to healthy patients (10–34 mm). Our patient's aortomesenteric distance was measured to be approximately 4 mm. Furthermore, the measured angle between the superior mesenteric artery and the aorta is reduced in patients with SMA syndrome from a normal range of 28°–65° to a measurement between 6°–22°. Our patient's aortomesenteric angle was difficult to measure secondary to poor sagittal reconstructions, but appears to be approximately 30°. Following radiographic evidence suggesting SMA syndrome together with our patient's constellation of presenting symptoms, a diagnosis of SMA syndrome was made and the patient was admitted to the general surgery service. However, our patient decided to leave against medical advice owing to improvement of his symptoms following the emptying of two liters of gastric contents via nasogastric tube evacuation.
[West J Emerg Med. 2012;13(6):501-502]

A 54-year-old male presents with a chief complaint of frequent vomiting for 20 hours after drinking alcohol. Previous medical history was significant for peptic ulcer disease status post perforation and surgical repair 1 year ago. On exam, vital signs were within normal limits. Physical exam revealed a distended abdomen with diffuse guarding and tenderness. Laboratory studies were within normal limits. A computed tomography (CT) of the abdomen and pelvis was ordered to further evaluate the etiology of the patient's symptoms revealing a severely distended stomach and distal duodenum with obstruction at the level of the superior mesenteric artery. These findings are consistent with superior mesenteric artery syndrome.

Superior mesenteric artery syndrome (SMA syndrome) is the result of compression of the third portion of the duodenum between the superior mesenteric artery and the abdominal aorta. Radiographically, SMA syndrome is characterized by several findings; compression of the duodenum between the abdominal aorta and superior mesenteric artery (Figure), dilation of the left renal vein, and distension of the stomach. In normal patients, the distance between the aorta and SMA (aortomesenteric distance) is 10–34 mm with aortomesenteric angle of 28°–65°.[1] Our patient had an aortomesenteric distance of approximately 4 mm (2-8 mm is common in patients with SMA syndrome) with an aortomesenteric angle of approximately 30° (Figure).[1] Most cases occur in patients with weight loss due to a variety of reasons (surgery, malabsorption, trauma, etc.) reducing superior mesenteric fat stores.[2,3,5] Common presenting symptoms include post-prandial abdominal pain, anorexia, nausea,

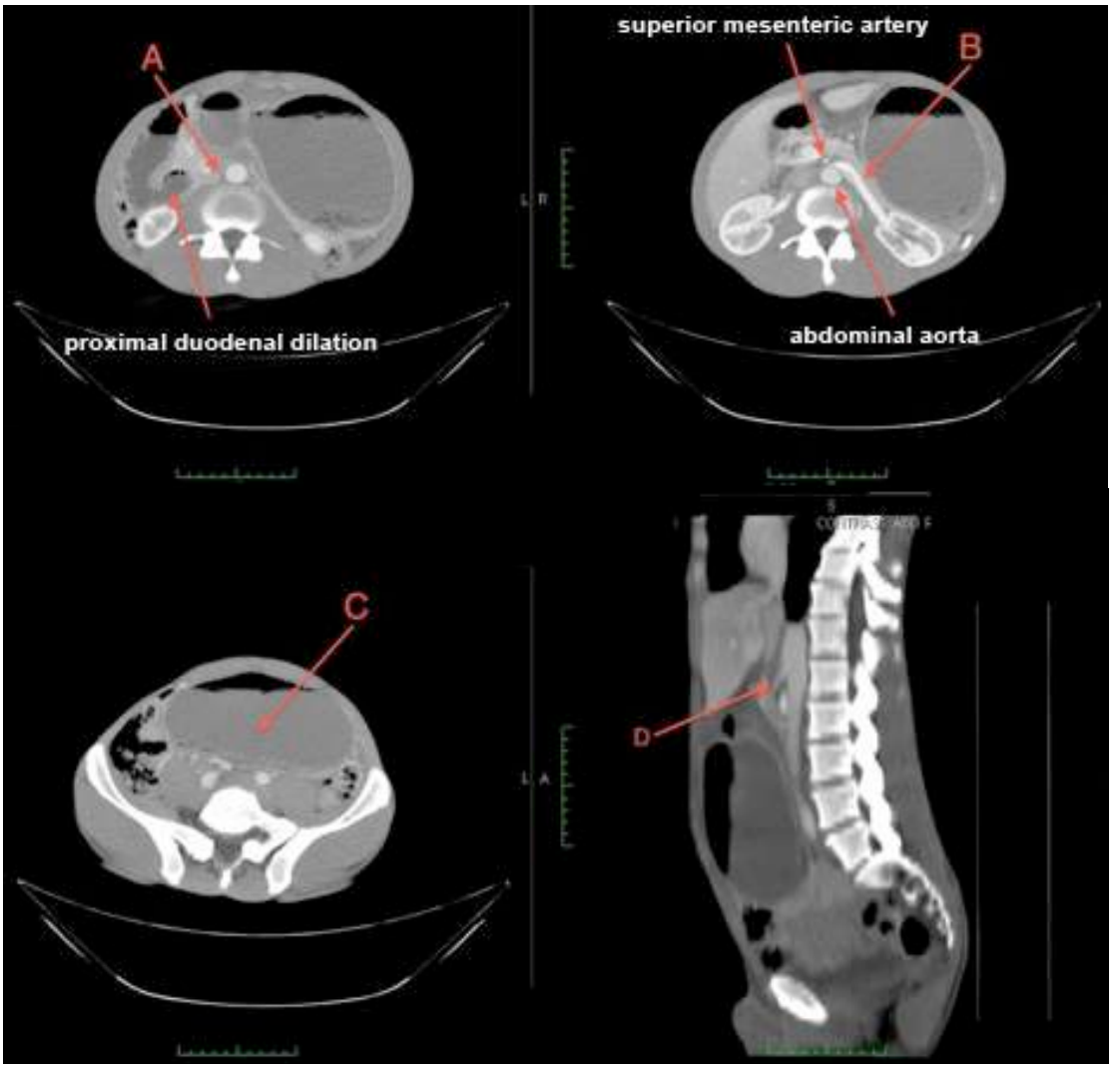

Figure. Computed tomography of the abdomen and pelvis demonstrating compression of the duodenum between the abdominal aorta and superior mesenteric artery (A), dilation of the left renal vein (B), distension of the stomach (C), and the aortomesenteric angle (D).

emesis and subsequent weight loss.[2-5] Surgical interventions (duodenojejunostomy most commonly) are employed if conservative measures fail.[2,3] Our patient underwent nasogastric tube placement with suction resulting in evacuation of 2 liters of gastric contents and was admitted to general surgery only to leave against medical advice 4 hours following admission as his symptoms had resolved.

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One in a Million: A Case of Arm and Leg Pain and Deformity

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A 38-year-old Hispanic woman with no known past medical or family history presented to the emergency department with severe, intractable left upper and lower extremity pain and inability to walk for 2 days. The woman reported a history of chronic, progressive left hand, arm, and leg deformity over the previous 2 years with episodic flares of severe pain. The woman had not reported any trauma or systemic symptoms. Physical exam revealed deformity and hypertrophy of the second and third digits of the left hand (Figure 1), the left elbow, left thigh, and lower aspect of her left leg without significant joint swelling, warmth or redness. She exhibited significantly limited and painful range of motion at the joints in the left upper and lower extremity. Plain radiographs were obtained (Figure 2).

DIAGNOSIS

Melorheostosis was first described by Leri and Joanny in 1922.[1] It is a rare osteosclerotic bone dysplasia caused by a non-inheritable, developmental error in the LEMD3 gene.[2] Its incidence is about 0.9 per million and effects males and females equally.[3] The pattern of the dysplasia generally follows a sclerotomal distribution, representing the zones of the skeleton supplied by individual spinal sensory nerves, and is typically asymmetric involving the extremities and rarely the axial skeleton. The name melorheostosis is derived from its characteristic "melting wax flowing down a candlestick" appearance on radiologic exam.[3]

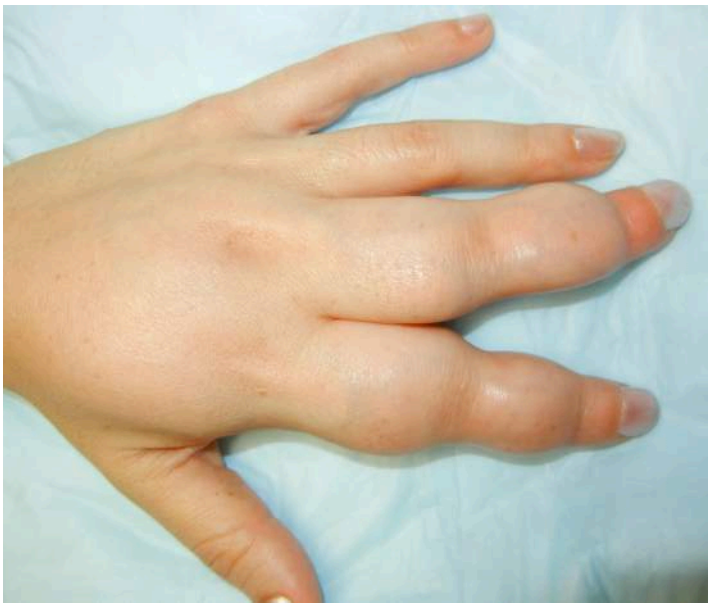

Figure 1: Photograph of the patients left hand demonstrating marked deformity and apparent swelling of the 2nd and 3rd digits and metacarpal bones.

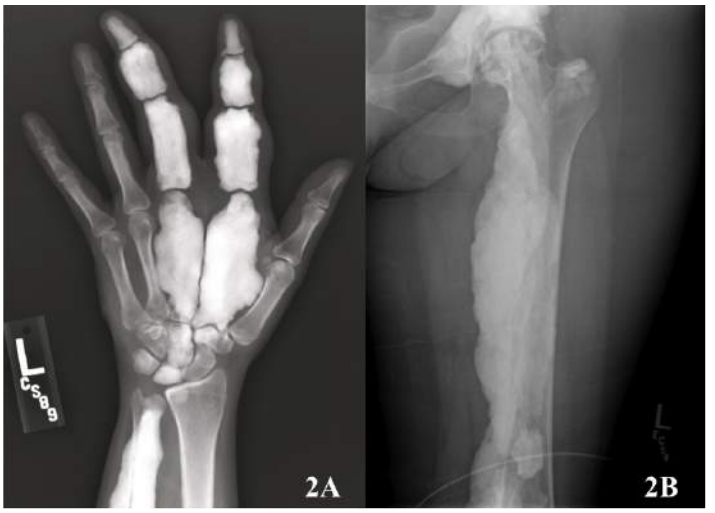

Figure 2: (A) Radiograph of the left hand showing dense sclerosis, lengthening, and expansion of the second and third phalanges and metacarpals with increased density of the lunate, capitate, and trapezoid and, (B) radiograph of the left proximal femur showing diffuse dense cortical thickening with characteristic "flowing candle wax" appearance.

Common presenting symptoms include pain, edema, stiffness, and deformity. The treatment of melorheostosis includes symptomatic treatment for pain, surgery to correct deformities, and amputation if necessary.

Our patient was admitted to the hospital for pain management. Two days later she was discharged to a skilled nursing facility for continued physical therapy, occupational therapy, and medical management.

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Severe Neck Pain with Fever: Is it Meningitis?

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A 58-year-old male patient presented to the emergency department with complaints of severe neck pain. He admitted to drug use but denied using intravenous (IV) drugs. On exam, he had a fever of 100.7°F, positive Kernig's sign, and normal neurologic exam. The patient was suspected to have bacterial meningitis and was started on IV antibiotics. The next day the patient developed decreased hand grip. Magnetic resonance imaging of the spine the next day showed a soft-tissue mass impinging on the spinal canal. The patient was subsequently taken to the operating room where the epidural abscess was drained. [West J Emerg Med. 2012;13(6):505–506.]

INTRODUCTION

Epidural spinal abscess is a dangerous spinal infection that can lead to compression or ischemia of the spinal cord with devastating neurologic outcomes. Patients often present with back pain and fevers.[1] Up to half of patients may present with no neurologic deficits. *Staphylococcus aureus* is the most common organism identified in epidural abscesses.[2] Patients at high risk for abscess include intravenous (IV) drug users; patients with alcoholism, human immunodeficiency virus, or diabetes; patients on long-term steroids; patients who have had recent bacterial infections; or patients with indwelling catheters.[3] Treatment includes antibiotics and neurosurgical consultation for operative drainage. Early recognition is key. However, only 15% of patients have cervical involvement; thus, these patients are often misdiagnosed in the emergency department (ED).

CASE PRESENTATION

A 58-year-old man presented to the ED after 2 days of body aches and neck pain. He described the neck pain as 10 on a scale of 10 (with 10 being high) and spasm-like with radiation to his upper arms. The patient also admitted to having a chronic nonproductive cough. On review of systems, the patient was found to have urinary frequency, generalized weakness, and abdominal pain. He had also had a 10-lb weight loss in the previous 2 weeks. The patient denied having fevers, chills, trouble breathing, chest pain, headaches, nausea, vomiting, or diarrhea. The patient had been hospitalized the previous week because of exacerbation of his chronic obstructive pulmonary disease (COPD) and underlying pneumonia.

The patient had a medical history pertinent for COPD and had undergone multiple intubations in the past. He was on albuterol and prednisone at home. He denied having any allergies. The patient had undergone 1 hernia surgery in the past. He smoked half a pack of cigarettes per day and used intranasal cocaine and heroin. He denied alcohol use or IV drug use.

On presentation to the ED, the patient was awake, alert, and in no acute distress. Vital signs included a temperature of 100.7°F, heart rate of 116 beats/minute, blood pressure of 135/62 mmHg, respiratory rate of 18 breaths/minute, and oxygen saturation of 98% on room air. The head, eye, ear, nose, and throat exam was negative. The patient had cervical spinal tenderness and a positive Kernig's sign. He also had intermittent wheezes on lung exam. Results of the heart exam were normal. The patient also had right upper quadrant tenderness with a negative Murphy's sign, no guarding, and no peritoneal signs. The neurologic exam was normal for motor, strength, and cranial nerves.

Laboratory results included a white blood cell (WBC) count of 20.1 and sodium levels of 134 mEq/L. Lactate levels were normal. Lumbar puncture showed 634 cells/μL WBCs with 93% neutrophils and 83 red blood cells. Cerebrospinal fluid protein levels were elevated at 366 mg/dL, and glucose was 60 mg/dL. Computed tomographic examination of the brain was negative for any acute process.

Because of the results of the lumbar puncture, the patient

was suspected to have bacterial meningitis. The patient was started on dexamethasone, vancomycin, ampicillin, and rocephin. The infectious disease department was contacted, and the patient was also given cefepime and acyclovir because of his recent hospitalization and drug use.

On admission, the patient continued to spike fevers and have pain in his neck. Blood cultures showed gram-positive cocci in clusters. On neurologic exam, the patient was now found to have decreased strength in his hands and decreased motor function in his bilateral distal extremities. Magnetic resonance imaging (MRI) of the spine showed a left paracentral soft-tissue mass thought to be an epidural abscess that was impinging on the spinal cord and was suggestive of anterior cord compression with focal edema.

The next day, a neurosurgeon performed a C2 to C3 epidural fluid collection and drainage. The patient was then continued on IV antibiotics for 6 weeks. The patient continued to deny IV drug use.

DISCUSSION

A spinal epidural abscess is a collection of pus or granulation tissue in the epidural space. The most common predisposing factors for abscess include diabetes and IV drug use, although there are case reports of patients with cervical spinal abscess and no predisposing factors.[4] Back pain and fever are common physical exam findings. The cervical spine is the least affected area.[5] When treated conservatively with antibiotics alone, patients with an epidural spinal abscess tend to deteriorate.[6] When the diagnosis is made, therefore, prompt surgical referral and operative debridement may lead to better outcomes. The preferred test for diagnosis is MRI with gadolinium.

The patient's physical exam findings on presentation and results of the lumbar puncture pointed to a diagnosis of meningitis. However, results of neurologic testing throughout the patient's hospital stay, combined with his recent hospital stay and therefore possible risk factor for sepsis, led to the diagnosis of cervical spinal abscess.

CONCLUSION

Epidural spinal abscesses are rarely reported in the cervical neck region and are often misdiagnosed as meningitis or another pathological condition. Early recognition is key, followed by appropriate antibiotic coverage and emergent neurosurgical intervention. Diagnosis is made with MRI with gadolinium of the spinal column. In patients presenting with neck pain, fevers, neurologic signs, and risk factors for abscess, prompt diagnosis is important for better outcome.[3]

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Asterixis as a Presentation of Cerebellar Ischemic Stroke

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Asterixis is not yet considered a common neurological sign of cerebellum infarction, and the pathogenic mechanism for asterixis remains elusive. We report a 58-year-old male with moderate hypertension who presented to our emergency department for acute headache in both cervical and occipital regions of the left side. About 2 hours later the patient developed ipsilateral asterixis in the upper left limb; 3 days later the asterixis disappeared. Magnetic resonance imaging of the brain disclosed cerebellar infarctions at the left superior cerebellar artery. In conclusion, we observed that a transitory asterixis associated with ipsilateral headache can be an initial clinical manifestation of ipsilateral cerebellar infarctions in the superior cerebellar artery area. [West J Emerg Med. 2012;13(6):507-508]

INTRODUCTION

Involuntary movements, such as chorea, dystonia, asterixis, and tremor, may occur as a consequence of stroke due to the involvement of basal ganglia or thalamus/subthalamus.[1,2] Involuntary movements caused by the anterior cerebral artery infarction have been reported.[3] While asterixis has been described in 2 patients with ipsilateral cerebellar infarction, it is not yet considered a neurological sign of cerebellum infarction.[2] We report a patient with unilateral headache and transitory asterixis of the upper left limb due to an acute cerebellar infarction in the area of the superior cerebellar artery.

CASE REPORT

A 58-year-old male with a history of moderate hypertension and treatment with enalapril (20 mg/day) presented to our emergency department with acute headache in both the cervical and occipital regions of left side. About two hours later he developed asterixis of the upper left limb and gait instability. There was no history of alcohol or drug abuse, and no significant past medical or surgical history. History did not reveal symptoms consistent with depression and/or anxiety, and the patient was only taking enalapril. Neurological examination showed gait instability. Biochemical tests, including serum ammonia, ceruloplasmin, and hematological tests, were normal. Blood pressure was 140/80 mmHg and electrocardiogram showed a sinus rhythm (72 beats/min). Baseline electroencephalogram disclosed mild, generalized, background alpha activity. Ultrasound of the neck was normal. Computer tomography of brain did not reveal structural lesions or abnormalities. The hyperkinetic movement disorder diminished in intensity and was observed as a pattern of asterixis only when the hands were stretched. Over the next 3 days, the asterixis disappeared and neurological examination revealed a weakness in the upper left limb (IV/V on the 0-V Medical Research Council scale). Magnetic resonance imaging (MRI) of the brain disclosed cerebellar infarctions in the left superior cerebellar artery region (Figure). Because angio-MRI did not reveal abnormality, we started the patient on aspirin (100 mg/day). Five days later, clinical examination revealed a weakness in the upper left limb (IV/V) with slight gait instability and the patient was discharged. Clinical evaluations performed one month later did not reveal any signs of neurological lesions.

DISCUSSION

Asterixis may be induced by a focal structural brain lesion such as a stroke and involves the midbrain, thalamus, parietal lobe, or frontal cortex.[1,2,5] The incidence of post-stroke asterixis remains unknown. Previous studies reported bilateral

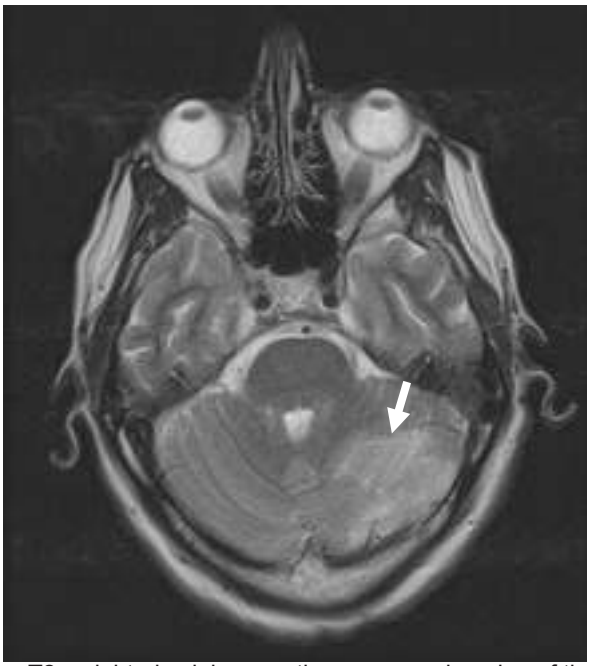

Figure. T2-weighted axial magnetic resonance imaging of the brain disclosed cerebellar infarctions in the left superior cerebellar artery region (arrow).

asterixis due to unilateral brain lesions after infarction in the anterior cerebral artery region.[2,5] In our patient the asterixis started immediately after the onset of stroke at the superior cerebellar artery and resolved within 3 days. The pathogenic mechanism for asterixis remains elusive. Previously it has been reported that asterixis is a negative myoclonus caused by intermittent failure in maintaining sustained muscle contraction.[1,2] Both the anatomic location and the presence of gait instability in our patient suggest that asterixis may reflect a failure in arm posture maintenance, inducing the failure in the leg posture control. The postural stability or tonic control of the extremities is related to multiple brainstem–spinal pathways, such as the vestibulospinal, reticulospinal, and rubrospinal tracts. These systems are regulated by supratentorial structures; the ventral lateral nucleus of the thalamus is the area where the cerebellar–rubral or vestibulocerebellar fibers converge, and it is also strongly connected with the prefrontal area.[4] The projections from the medial frontal cortex to the brainstem reticular formation may play a role in the regulation of muscle tone or posture.[4] The occasional occurrence of bilateral asterixis and the transient nature of symptoms suggest that the system regulating posture maintenance is not strictly unilateral.

In a clinical study the author reported that the occurrence of ipsilateral asterixis in patients with cerebellar lesions can be explained by crossing cerebellar–rubral fibers at the superior cerebellar peduncle.[4] Our patient's asterixis was ipsilateral to acute cerebellar infarction, confirming that finding.

In conclusion, we reported that a transitory unilateral asterixis associated with ipsilateral headache can represent an initial clinical manifestation of ipsilateral cerebellar infarcts in the region of the superior cerebellar artery territory.

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Subdural Empyema Presenting with Seizure, Confusion, and Focal Weakness

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While sinusitis is a common ailment, intracranial suppurative complications of sinusitis are rare and difficult to diagnose and treat. The morbidity and mortality of intracranial complications of sinusitis have decreased significantly since the advent of antibiotics, but diseases such as subdural empyemas and intracranial abscesses still occur, and they require prompt diagnosis, treatment, and often surgical drainage to prevent death or long-term neurologic sequelae. We present a case of an immunocompetent adolescent male with a subdural empyema who presented with seizures, confusion, and focal arm weakness after a bout of sinusitis. [West J Emerg Med. 2012;13(6):509-511]

INTRODUCTION

Intracranial suppurative lesions are life-threatening surgical emergencies that are rarely seen, difficult to diagnose, and can have serious long-term morbidity if not diagnosed and managed promptly and appropriately.[1,2] When present, these lesions are found often in adolescents and can present with non-specific symptoms of fever and headache. While meningitis and encephalitis are often the first diagnoses considered in this case, space-occupying lesions such as abscesses or subdural empyemas must be considered as well, in particular when there are neurologic deficits, altered mental status or seizures at presentation. We present a case of an adolescent male who presented with confusion, seizure, and left arm weakness due to a subdural empyema.

CASE REPORT

A 16-year-old male presented to the ED with 1 day of a reported fever, a right temporal headache, and a witnessed tonic-clonic seizure. He arrived confused with left arm weakness noted as well. He had no past history of seizures. For the preceding week, he had complained of sinus congestion and received an antibiotic from his primary care provider several days prior.

His review of systems was negative for rash, emesis or dyspnea. His medical history was unremarkable, and his immunizations were up to date. He lived at home with his family, none of whom were ill, and he took no medications or over-the-counter medications. He denied any allergies. Physical examination revealed a drowsy, but arousable, black male who was only oriented to person. His vital signs included a temperature of 36.6°C, pulse of 108, respiratory rate of 18, blood pressure of 118/54 mmHg, and an oxygen saturation of 100% on room air. His head and neck examination demonstrated only rhinorrhea but no meningismus, sinus tenderness, nystagmus or pupillary irregularity. His neurologic examination revealed isolated 0/5 weakness in the left upper extremity and confusion to time and place. His leg strength and reflexes were normal, but his gait was not tested. The remainder of his examination was normal. Diagnostic testing included an initial computed tomography (CT) that showed a right, frontal subdural hypodensity. His complete blood count was remarkable only for a white blood cell count of 17,300 cells/mm with a left shift. His basic metabolic panel showed normal electrolytes, glucose, and renal function. A magnetic resonance imaging (MRI) was obtained showing right frontal sinusitis with right subdural space extension (Figure). A subsequent lumbar puncture demonstrated no organisms on gram stain and 3 white blood cells per high power field.

He was diagnosed with a subdural empyema and started on intravenous ceftriaxone and vancomycin. He received supportive care and pain control, and he was admitted to the pediatric intensive care unit (PICU) with neurosurgical consultation. His empyema was surgically drained on the day of presentation. The patient slowly recovered in the PICU without any further seizure activity. At the time of discharge, his left arm strength was 4/5 and he had a normal mental status. He was

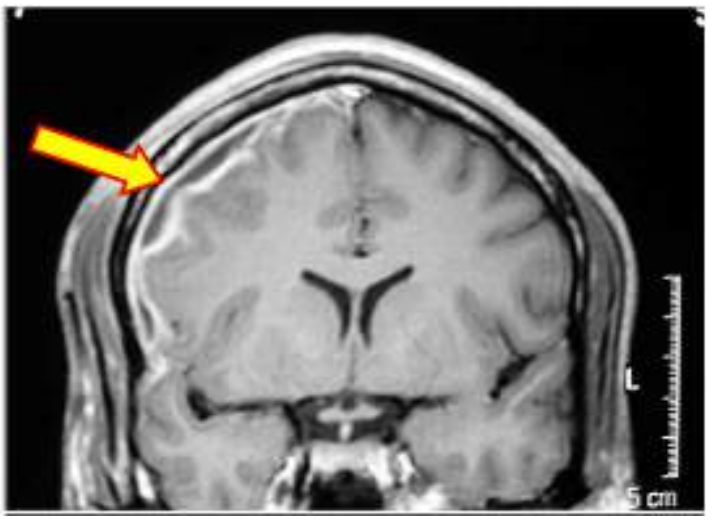

Figure. Magnetic resonance imaging revealing a right subdural empyema with meningeal enhancement.

kept on anti-seizure prophylaxis for 6 months without recurrent seizures, and his strength returned to normal with physical therapy over the subsequent 6 months.

DISCUSSION

Subdural empyemas are neurosurgical emergencies that require prompt recognition and management to salvage a good functional neurologic outcome. These lesions progress rapidly and cause increased intracranial pressure leading to coma and death within 24 to 48 hours if untreated.[2,3] Historically, these rare complications occurred secondary to otitis media, but currently they are seen more frequently as a complication of bacterial sinusitis.[4] A subdural empyema is a known but rare suppurative complication of bacterial sinusitis. The suppurative complications of sinusitis can be divided into two categories – orbital and intracranial. Orbital complications classically occur from infection of the ethmoid air cells and can include orbital cellulitis, orbital abscesses, and subperiostial abscess (Pott's puffy tumor). Intracranial complications include subdural empyema, epidural abscess, intracerebral abscess, meningitis, and thrombosis of the cavernous and other dural sinuses. Subdural empyema is the most common intracranial complication associated specifically with sinusitis.[3,5] Historically, subdural empyema was the most common intracranial suppurative complication overall, but epidural abscess may now be the more common entity.[6] However, of the possible suppurative intracranial complications, subdural empyemas have the worst long term outcomes.[3] There are two potential mechanisms for subdural empyema formation following a bout of bacterial sinusitis. First and most commonly, a retrograde thrombophlebitis occurs via the valveless diploic veins and seeds the subdural space. Second, direct extension can occur from an infected sinus. Frontal sinusitis is the most common culprit in this instance. Close anatomic proximity of the sinus to the subdural space allows for this direct extension. The infection may also erode the facial bones causing osteomyelitis and subsequent direct extension as well. Once the infectious process invades the subdural space, a rapid clinical deterioration is common.[3,6] Subdural empyemas are often polymicrobial. The most common pathogens are micoraerophilic and anaerobic gram positive cocci, but anaerobic gram negative bacilli may be present as well.[7] The most commonly isolated organisms are from the *Streptococcus milleri* family, but the type of organism isolated has not been associated with the severity of disease or the outcome.[3,6,8] A preceding course of oral antibiotics may make isolating the offending pathogen difficult, and blood cultures are rarely positive for the infecting bacterium.[8] As the use of antibiotics has become more prevalent over the past few decades, the incidence of sinogenic brain abscess has decreased.[9] Subdural empyemas present most commonly in the second and third decades of life.[3] While they can have a wide variety of symptoms, fever and progressive headache are the most common.[2] Some patients do not have a known history of sinus disease, and others may have chronic or subacute sinusitis which can confound the clinical diagnosis further. [3,5,6,8] Germiller et al[6] reported a mean symptom duration of 12 days prior to diagnosis, and Farah and colleagues[10] found symptoms took 2-6 weeks to develop from sinusitis to signs of intracranial infection. This subacute course and vague symptoms contribute to the risk of misdiagnosis or delay in diagnosis.[10] Altered mental status, seizures, and hemiparesis are the most common neurologic manifestations, but focal neurologic deficits may be present as well, particularly if there is an associated intracranial abscess. Interestingly, intracranial abscesses occur concomitantly in 6-22% of cases.[3] Neurologic findings are particularly ominous and vary depending on the location of the lesion, but when they are present, rapid neurosurgical intervention is necessary because these symptoms will worsen rapidly.[3] Other nonspecific symptoms include nausea, vomiting, meningismus, personality changes, papilledema, periorbital edema, and cranial nerve palsies.[5] Because many of these patients may present with vague symptoms, if there are extracranial symptoms, such as facial swelling, periorbital edema, or proptosis, or ocular findings such as vision loss, decreased or painful extra-ocular motion, and diplopia, further testing is recommended because extracranial symptoms may be a harbinger of an intracranial infectious process.[5] Singh et al[11] found that 37% of patients with subdural empyemas had concomitant extracranial complications. Finding these extracranial complications before neurologic symptoms have developed will improve the likelihood of an uncomplicated outcome.[6,8]

In this era of frequent CT use, early diagnosis is much more likely to occur than previously, and this has likely contributed to the decreased mortality and long-term morbidity of suppurative intracranial infections. Imaging studies should be performed immediately if there is concern for intracranial extension. CT is typically the first-line choice because it is readily available

and may show edema or mass effect. CT should be done with intravenous contrast if there is concern for an intracranial infection. If it is negative, and a subdural empyema is still considered, an MRI must be obtained with gadolinium contrast. MRI is the most sensitive test for intraparenchymal and intracranial infections and is considered the gold standard.[5,7] One recent study found CT to have a 63% sensitivity for sinogenic intracranial complications while MRI was 93% sensitive.[6] Management of subdural empyemas includes immediate administration of a third generation cephalosporin plus metronidazole and vancomycin to provide appropriate coverage against streptococci species and *Staphylococcus aureus*.[5,7] Adjunctive seizure prophylaxis is recommended early as well, as seizures may be present in up to 20% of cases. [3,8,12] Interventions to lower intracranial pressure (elevation of the head of the bed, mannitol, and ventriculostomy) should be performed as necessary. Emergent neurosurgical consultation is paramount as surgical intervention (burr hole or craniotomy) is necessary in most cases to provide the best opportunity at neurologic recovery.[2] Likewise, otolaryngologic consultation is recommended because functional endoscopic sinus surgery (FESS) may aid in drainage and recovery.[5,8] Historically, mortality from subdural empyema has been as high as 15-41% even with surgical drainage.[3,10,13] Antimicrobials have significantly lowered the mortality rate, as subdural empyema was universally fatal prior to antibiotic use.[3] The advent of CT and MRI has also helped to decrease the mortality rate, with recent studies suggesting a mortality rate between 6% and 15%.[3,14-17] Germiller and colleagues[6] reported a 4% mortality rate in a pediatric series of intracranial suppurative infections. Survivors often have significant morbidity with 12-37.5% having persistent seizures, hemiparesis in 15-35% , or residual neurologic deficits in up to 50% of survivors.[3,14-16]

CONCLUSION

Intracranial suppurative complications, such as subdural empyemas, are rare and difficult to diagnose because initial symptoms may be vague. Patients with sinus infections and progressive headaches or any neurological deficits should be aggressively evaluated with CT or MRI imaging as needed to rule out intracranial spread. Because subdural empyemas have serious morbidity and mortality if not recognized and treated promptly, emergency physicians must be aware of the risks of subdural empyema and the need to emergency neurosurgical intervention.

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Electrocardiogram changes in Thyrotoxic Periodic Paralysis

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Thyrotoxic periodic paralysis (TPP) attacks are characterized as recurrent, transient episodes of muscle weakness that range from mild weakness to complete flaccid paralysis. Episodes of weakness are accompanied by hypokalemia, which left untreated can lead to life-threatening arrhythmias (6). In this case study, we followed a patient's potassium levels analyzing how they correlate with electrocardiogram changes seen while treating his hypokalemia and ultimately his paralysis. [West J Emerg Med. 2012;13(6):512-513]

INTRODUCTION

The overall incidence of thyrotoxic periodic paralysis (TPP) is 0.1-0.2% in North America, but varies dramatically among certain populations.[1] In Chinese and Japanese patients, the incidence is 1.8-1.9%, the highest among any one population.[2] Attacks are characterized as recurrent, transient episodes of muscle weakness that range from mild weakness to complete flaccid paralysis. Episodes of weakness are often accompanied by hypokalemia, and treatment of the underlying thyrotoxicosis will resolve symptoms of muscle weakness.[3] Aggressive replacement of potassium can cause a drastic rebound hyperkalemia, but left untreated, severe hypokalemia can also cause life-threatening arrhythmias.[4-6]

In this case, we report the presence of 3 distinct arrhythmias in a single patient as his potassium levelchanged during an episode of acute TPP.

CASE

A 29-year-old Asian male presented to the emergency department (ED) with symmetric paralysis of his lower extremities and weakness of his upper extremities that developed overnight. He had been recently diagnosed with hyperthyroidism 10 days prior, after presenting with 4 months of palpitations, muscle pain, cramping, and stiffness. He was started on propranolol and methimazole at that time. After resolution of his original symptoms, he stopped taking the medications and presented to the ED with complete paralysis of his lower extremities.

On further history, it was discovered that over the preceding 4 months the patient had clinical features of TPP that had been subtle, with reports of transient lower extremity weakness occurring that resolved without medical intervention.

On physical exam, the patient had symmetrical weakness and was unable to move his legs, but able to move his upper body, with sensation still intact. Upon initial laboratory evaluation, the basic metabolic panel (BMP) demonstrated a potassium level of 1.7 milliequivalent (mEq/L). The free thyroxine (T_3) level was elevated at greater than 8.8 ng/dL, and thyroid-stimulating hormone (TSH) was decreased at

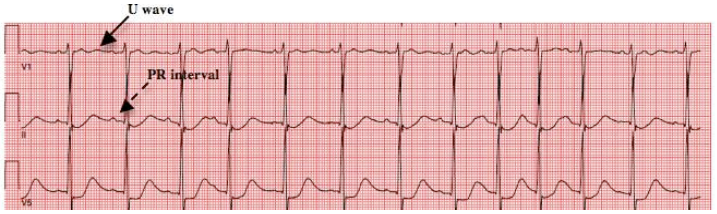

Figure 1. The patient's potassium was initially 1.7 meq/L. The PR interval is prolonged and prominent U waves are present.

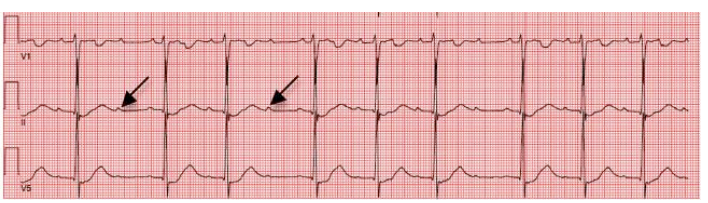

Figure 2. This electrocardiogram shows Wenckebach or second degree heart block, Mobitz Type 1, which deveoped once the potassium dropped to 1.6 meq/ L. Solid arrow shows dropped beat.

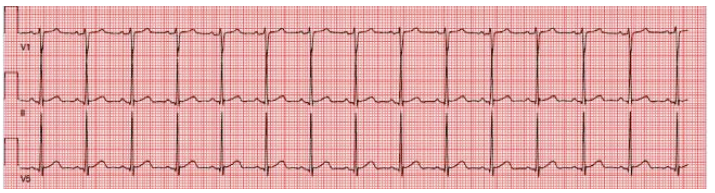

Figure 3. The electrocardiogram shows resolution of the patient's second degree heart block and U waves, with serum potassium of 3.1 meq/L.

0.010 µlU/mL. The initial electrocardiogram (ECG) showed 1st degree heart block with prominent U waves (Figure 1). The PR interval was prolonged to greater than 0.2 sec as seen in leads V1 & V5, and U waves were present in all leads. There were no prior ECGs to compare these findings. Treatment was initiated with slow potassium repletion, but the potassium continued to drop to 1.6 mEq/L and the ECG showed 2nd degree heart block, Mobitz Type I, best seen in Lead II and V1 (Figure 2).

The development of this 2:1 AV block coincided with increased weakness of more proximal muscles and decrease in reflexes. However, the patient's airway was never compromised because his respiratory muscles were always intact. The paralytic attack was aborted with a combination of cautious potassium replacement, methimazole and parenteral propranolol.The patient received initial doses of potassium chloride 10 mEq intravenously and 40 mEq orally. A repeat BMP 3 hours later revealed a potassium level of 3.10 mEq/L and magnesium level of 1.8 mmol/L. Subsequently, the PR interval was noted to shorten and the rhythm returned to normal sinus (Figure 3).

Our patient exhibited numerous ECG changes due to hypokalemia secondary to thyrotoxicosis, with associated thyrotoxic periodic paralysis. Symptoms resolved, along with the seen ECG changes, with cautious potassium repletion and control of the underlying thyrotoxicosis.

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Thyroid Evaluation in a Patient with Thyrotoxicosis with Bedside Ultrasound

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A 37-year-old man presented to the emergency department (ED) for palpitations, anxiety, and weight loss. Physical examination revealed a pulse of 104 beats/min, blood pressure 140/92 mmHg, respirations 12 breaths/min, and oral temperature 99 F. Neck examination was notable for a diffusely enlarged thyroid without discrete nodules. Bedside emergency ultrasound (EUS) was performed with a high frequency 10 MHz linear array probe. This demonstrated an enlarged thyroid with a heterogeneous appearance, but no discrete nodules or masses. Color flow Doppler ultrasound showed profoundly increased blood flow to the thyroid (Video). These sonographic findings are consistent with a hyperthyroid state, most commonly due to Graves' disease.[1,2] Serum thyroid studies revealed a very suppressed TSH and an elevated free T3/T4. Based on the patient's ultrasound and laboratory testing, he was diagnosed with thyrotoxicosis. Treatment with propranolol and methimazole was initiated from the ED in consultation with Endocrinology. He was discharged with close clinic follow up.

In the ED it is important to diagnose thyrotoxicosis early in its course, since it can potentially progress to thyroid storm. While relatively uncommon and affecting only 10% of patients hospitalized for thyrotoxicosis, thyroid storm carries a mortality rate of 20-30%.[3,4] Furthermore, untreated thyrotoxicosis can cause or worsen hypertension, congestive heart failure, atrial fibrillation, metabolic disorders, osteoporosis and neuropsychiatric disorders.

Thyroid ultrasound is best performed using a high frequency probe, scanning through the gland in both transverse and vertical orientations. The normal thyroid appears homogenous, with a characteristic echogenicity. Color Doppler ultrasound is then used to estimate thyroid blood flow.

This case demonstrates how EUS can be used as a rapid and helpful tool in the diagnosis of thyrotoxicosis. An enlarged thyroid gland with hypervascular flow on Doppler ultrasound can corroborate clinical data and lead to the correct diagnosis in a timelier manner.

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Ultrasound Diagnosis of Urethral Calculi

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A 35-year-old man presented to the emergency department (ED) for acute urinary retention and penile pain for 4 hours. The patient denied any significant medical history or history of trauma. Physical exam revealed testicles that were nontender, without masses. However, a tender mass was felt at the distal end of the penis, adjacent to the urethral meatus. Placement of a Foley catheter resulted in a return of 700 cc of clear yellow urine and immediate resolution of the patient's suprapubic and penile pain.

During the ED course, the Foley catheter was removed with a subsequent trial of voiding. Initially, the patient was able to void 15 cc of urine until the normal stream was abruptly cut off. The patient then complained of extreme penile pain, near the urethral meatus. A small, circular and firm mass was again palpated in the distal penile shaft. Bedside emergency ultrasound (EUS), performed with a 10 MHz linear array probe placed along long axis of penis, revealed a hyperechoic, dense and round structure with characteristic acoustic shadowing at the distal end of the urethra, with obstruction of the urinary flow (Video). The object, a 9 mm stone, was removed with forceps. Following stone removal, the patient experienced immediate pain relief and was able to spontaneously void.

While urethral imaging has traditionally been performed with retrograde urethrography (RUG), more recently ultrasound has been used to minimize the pain associated with RUG and to provide clinicians more detailed information about urethral pathology.[2] As demonstrated in this case, EUS allowed a prompt diagnosis of the patient's condition with appropriate rapid treatment and removal of the urethral stone.

Video. Ultrasound of dorsal surface of penis with 10 MHz linear transducer demonstrating urethral calculus at urethral meatus (audio narration included).

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Pulmonary Hypertension, Hemoptysis and an Echocardiographic Finding of a Ventricular Septal Defect

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A 63-year-old female with pulmonary hypertension presented to the emergency department (ED) with hemoptysis. Vital signs were significant for tachypnea, with respirations of 30 per minute, and hypoxia, with an oxygen saturation of 88%. Physical examination revealed a systolic cardiac murmur. Bedside emergency ultrasound (EUS) demonstrated a right ventricle that appeared to be enlarged in relation to the left ventricle, consistent with a diagnosis of pulmonary hypertension. In addition, a ventricular septal defect (VSD) was present. Color flow doppler showed a left to right shunt through the VSD (Video).

VSDs are the most common cardiac congenital defect at birth and account for 40% of all cardiac anomalies.[1] The normal directionality of blood flow across a VSD is left to right, due to the relatively higher pressure in the left ventricle. Eisenmenger syndrome is defined by the presence of systemic-to-pulmonary cardiovascular circulation, progressive pulmonary hypertension and increased right ventricular pressure. This leads to reversal of normal shunt flow with formation of a right-to-left shunt and resultant hypoxemia and cyanosis. Eisenmenger syndrome in association with VSD is often referred to as Eisenmenger complex.[2] Patients with Eisenmenger complex generally have poor outcomes due to increased strain on the heart, with shunting of oxygenated blood away from the core organs.[3,4]

This case details how EUS can aid the emergency physician in further evaluating the patient presenting to the ED with shortness of breath, hemoptysis and a cardiac murmur. In this case, a VSD with left-to-right shunt was diagnosed on EUS, simultaneously with right ventricular strain, underscoring the need for aggressive treatment of the patient's pulmonary disease due to the ominous possibility for the future development of Eisenmenger complex.

Video: Parasternal long axis view of the heart.

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Bladder Bulge: Unifying Old and New Sonographic Bladder Wall Abnormalities in Ureterolithiasis

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INTRODUCTION

Approximately 1% of all emergency department (ED) visits are for treatment of urinary tract stone disease (renal colic, kidney stones, urolithiasis).[1] Renal colic is a common condition affecting approximately 7–13% of the population during their lifetime and those who are afflicted are likely to have recurrent attacks throughout their lives.[2,3]

Noncontrast computed tomography (CT) of the abdomen and pelvis (CT KUB) is the gold standard imaging modality for urolithiasis. CT, however, is costly and exposes patients to potentially dangerous amounts of ionizing radiation, especially when performed repeatedly over time. Ultrasound (US) has been studied extensively in urolithiasis, specifically with regard to the finding of hydronephrosis as a secondary sign. Mentions of sonographic bladder wall abnormalities in renal colic, however, are rare in the literature and limited exclusively to a few brief descriptions of impacted uretovesicle junction (UVJ) stones.[4,5]

Through the following case series, we present sonographic bladder wall findings in patients with renal colic. These similar appearing abnormalities, which we propose to uniformly name the "bladder bulge," have not, to our knowledge, been previously discussed in the emergency medicine literature. One variant, in fact, may represent a novel sonographic description unto itself. The bladder bulge is easily obtained and can best be seen on axial views as a unilateral, inward bulging and/or focal thickening of the bladder wall on the symptomatic side, at the approximate level of the uretovesical junction (UVJ).

METHODS

We attempted to identify previous descriptions of bladder wall abnormalities in urolithiasis through a Medline search of English language journals using the following Medical Subject Heading (MeSH) terms and text words: *Ureteral calculi* or *renal calculi* or *urolithiasis* or *renal colic* or *ureterolithiasis* and combined with *ultrasonography* and *bladder*. Two hundred thirty-six articles were found. We then searched using only the keyword *pseudoureterocele*. Eight articles were found. We limited our search to English language, humans and adults. Of the first search, 2 articles briefly mentioned ureteral prolapse and edema as seen on US at the UVJ.[4,5] The second search yielded a single sonographic description of a pseudoureterocele in a patient with tuberculosis of the bladder.[6]

The following 7 cases presented to 2 different ED's between June 2011 and November 2011. These departments are part of general tertiary hospitals that include trainees in emergency medicine. The sonographers included 2 emergency medicine physicians who are fellowship-trained in emergency ultrasound and one emergency medicine trainee with limited formal training in ultrasound.

The patients were identified retrospectively through a search of the emergency department ultrasound (EDUS) logbook and image archive. All received both a bedside ultrasound performed by an emergency physician that included views of the bladder and affected kidney, and a CT KUB confirming the presence, location and size of the stone. We selected the axial US images most representative of the bladder bulge, together with the corresponding CT image, and presented them to a board certified radiologist for interpretation. The radiologist was blinded to the purpose of the project and given specific instructions to provide a highly detailed interpretation, describing as many aspects of the anatomy as possible, including elements that may not normally be mentioned in a standard reading. Only the portion of the reading relating to the bladder wall is included here. The radiologist also determined the presence or absence of hydronephrosis on EDUS.

Ultrasound Technique

Patients' bladders were thoroughly interrogated transabdominally in the axial plane using a low frequency (1–4 MHz), curvilinear probe. There was no standardization of bladder volume.

Cases

The following 7 patients, aged 28 to 58, presented with symptoms consistent with renal colic. The images show variations of the bladder bulge and are followed by our radiologist's interpretation. CT images are included where they were felt to represent the bladder bulge. Table 1 contains additional clinical variables.

Case 1: A 33-year-old male with a left UVJ stone

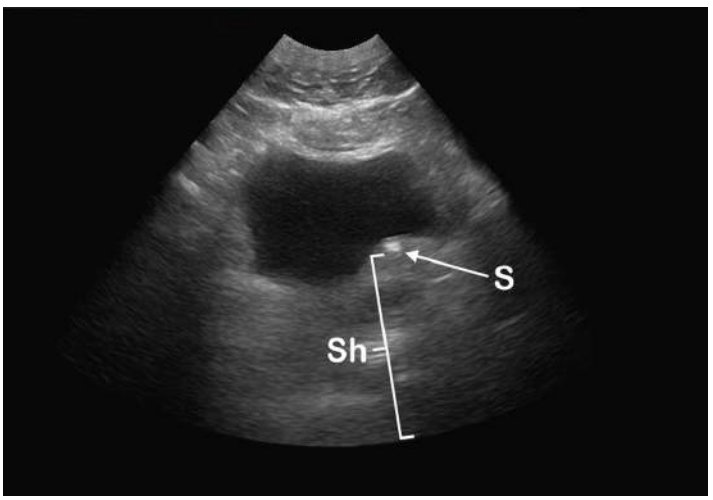

Figure 1. Emergency department ultrasound (EDUS). Direct stone visualization (S) with shadowing (Sh) and protrusion of the left posterior bladder wall.

Radiologist description: focal wall thickening and inward bulge along the left posterior wall, centered around a subcentimeter echogenic structure with shadowing, consistent with a distal ureteral calculus.

Case 2: A 33-year-old male with a right UVJ stone.

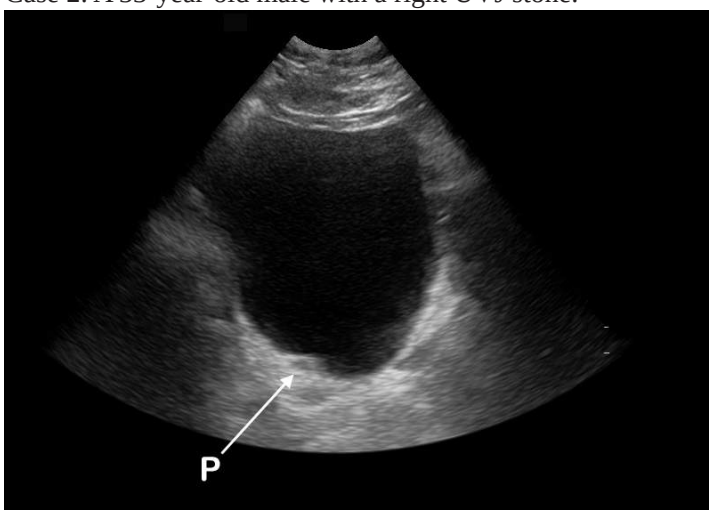

Figure 2. Emergency department ultrasound (EDUS). A pseudo-ureterocele (P) showing characteristic cystic appearance at the right posterior bladder wall.

Radiologist description: focal thickening of the right posterior bladder wall, with slight inward protrusion of this portion of the wall. There is also the suggestion of a small cystic focus at the level of the right UVJ, which may represent dilation of the intramural portion of the distal ureter.

Case 3: A 38-year-old male with a left UVJ stone

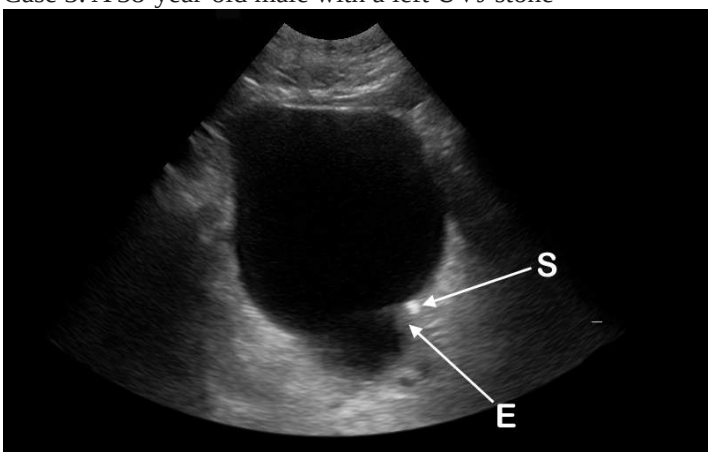

Figure 3. Emergency department ultrasound (EDUS). Direct stone visualization (S) with edema (E) and possible ureteric prolapse (P) at the left posterior bladder wall.

Radiologist description: Focal wall thickening around an echogenic calculus along the left posterior wall, with protrusion of the thickened wall into the bladder lumen.

Case 4: A 44-year-old female with a left ureteric stone (likely UVJ)

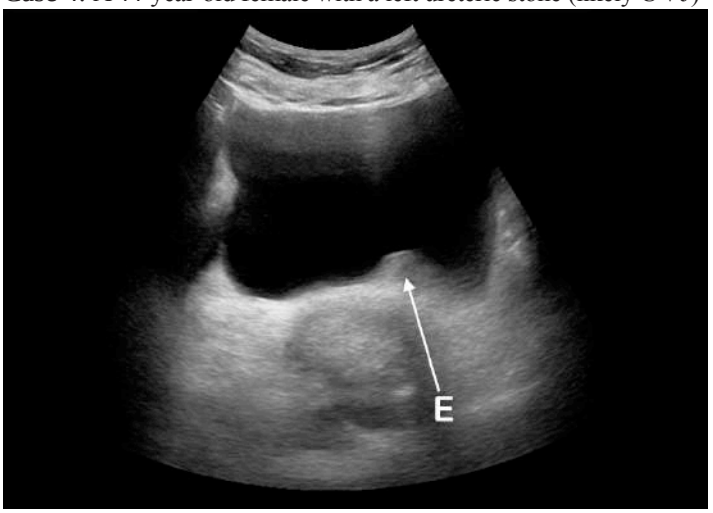

Figure 4. Emergency department ultrasound (EDUS). Inward protrusion from edema (E) without stone visualization at the left posterior bladder wall.

Radiologist description: inward protrusion of the left posterior bladder wall into the lumen.

Case 5: A 58-year-old man with a left distal ureteric stone (*not* near the UVJ).

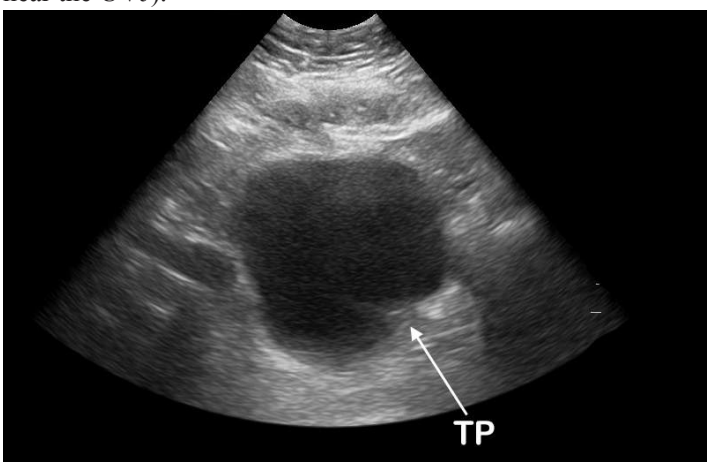

Figure 5a. Emergency department ultrasound (EDUS). Thickening (T) and inward protrusion (P) at the left posterior bladder wall.

Radiologist description: focal wall thickening and inward protrusion of the left posterior bladder wall.

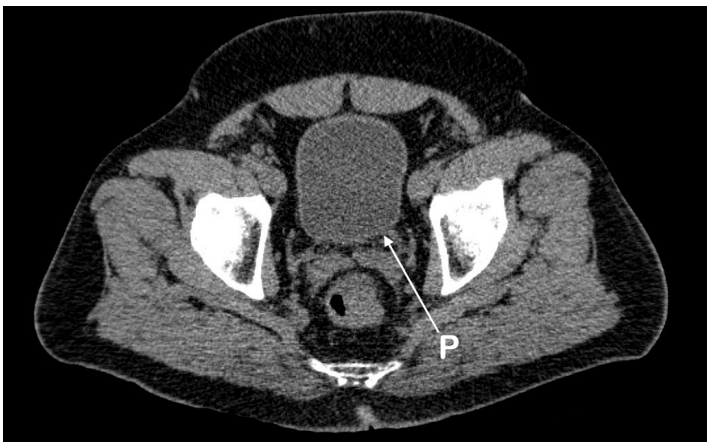

Figure 5b. Computed tomography (CT) of the abdomen and pelvis (CT KUB). Inward protrusion (P) of the left posterior bladder wall.

Radiologist description: minimal asymmetry along the posterior bladder wall, with an inward bulge along the left side.

Case 6: A 28-year-old male with a left proximal ureteric stone.

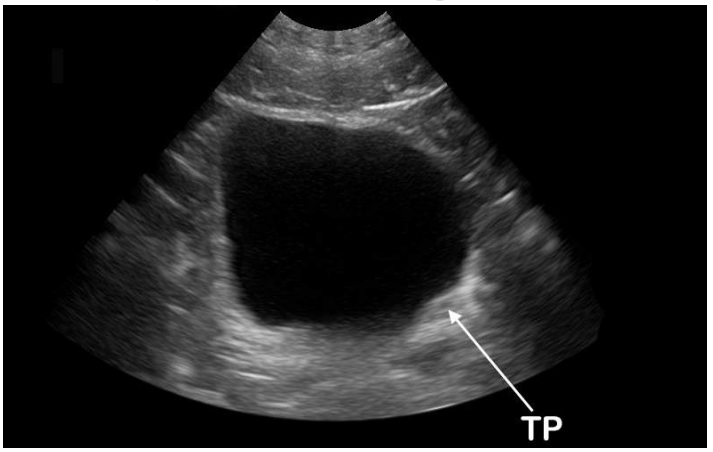

Figure 6. Emergency department ultrasound (EDUS). Thickening (T) and inward protrusion (P) of the left posterior bladder wall.

Radiologist description: irregularity of the bladder wall, with focal inward bulge and apparent thickening of the left posterior wall.

Case 7: A 44-year-old man with a left proximal ureteric stone

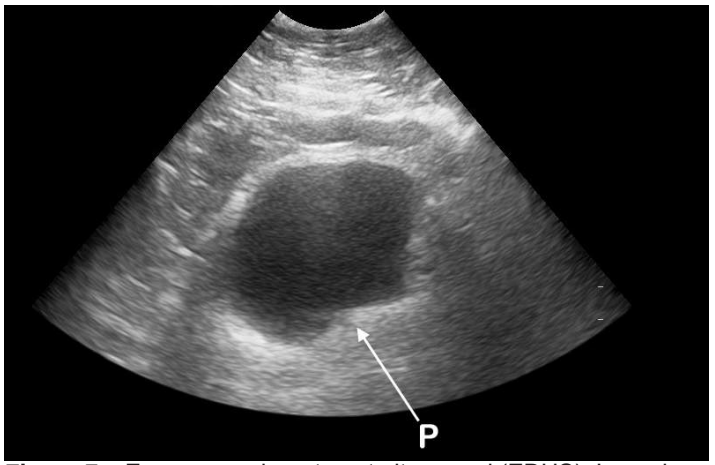

Figure 7a. Emergency department ultrasound (EDUS). Inward protrusion (P) of the left posterior bladder wall.

Radiologist description: minimal asymmetry along the posterior bladder wall, with flattening of the left side.

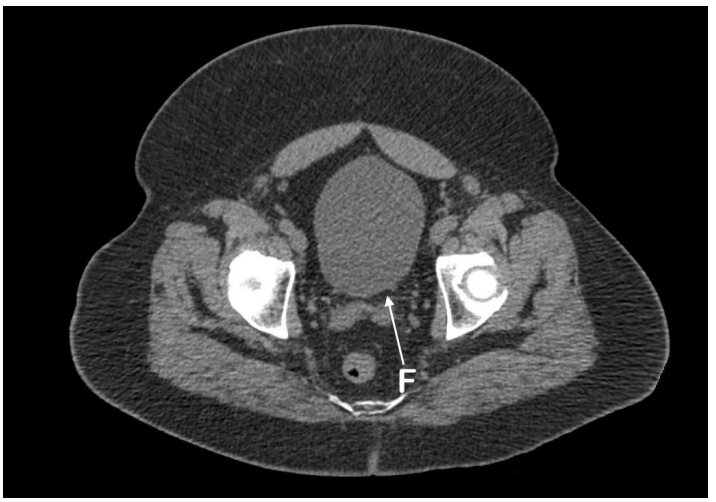

Figure 7b. Computed tomography (CT) of the abdomen and pelvis (CT KUB). Flattening (F) of the left posterior bladder wall.

Radiologist description: slight inward protrusion of the left posterior bladder wall.

All 7 patients in the case series had confirmed ureterolithiasis and an EDUS that showed a unilateral inward bulge of their bladder on the symptomatic side. Stone size and location varied (Table). In cases 5 and 7, there was the suggestion of a correlating bulge seen on CT (Figures 5a, 7a), although this finding was subtler. The bladder bulge sign is therefore felt to be primarily, if not exclusively, a sonographic finding. Case 4 had the CT performed 12 days prior to the EDUS making it likely that her stone had migrated to the UVJ by the time of her EDUS. Four of our patients had no hydronephrosis. One patient had neither hydronephrosis nor hematuria.

DISCUSSION

Renal colic is a painful, but generally benign condition with a hospital admission rate of 6–10%.[1,7] Fortunately, less than 10% of renal colic patients require intervention within 7 days and only 1–3% of patients undergoing CT KUB have an alternate diagnosis requiring emergent intervention.[1,7-9] Broder showed that of 262 patients who received a CT KUB for suspected renal colic, 244 (93%) had neither a need for urologic intervention nor an emergent cause for their symptoms.[7]

It is estimated that 1 CT abdomen/pelvis contains the equivalent radiation dose of 1000 single-view (posterior-anterior) chest x-rays, conferring an estimated lifetime cancer risk of 6:1000 in a 20-year-old.[10,11] Due to the nature of their disease, renal colic patients are likely to have multiple presentations to the ED and multiple CTs, as most emergency physicians, and even many radiologists, are not fully aware of the degree of radiation exposure.[12] To decrease their radiation risks, urolithiasis patients need an imaging modality that is harmless and effective.

Table. Additional clinical variables.

	Clinical Cases						
	1	2	3	4	5	6	7
Hematuria	Yes	Yes	Yes	Yes	Yes	Yes	No
US bladder volume	Moderate	Full	Full	Moderate	Moderate	Full	Low–Moderate
CT bladder volume	Full	Moderate	Full	Moderate	Full	Full	Full
Hydronephrosis on EDUS	Yes	No	No	Yes	No	Yes	No
Time from EDUS to confirmatory study (hrs:min)	5:00	-00:20	3:42	-12 days	5:00	2:35	24:29
Stone size	4 mm	5 mm	4 mm	7 mm	8 mm	6 mm	6 mm
Stone location	L UVJ	R UVJ	L UVJ	L PU	L DU	L UPJ	L DU
Bulge seen on CT	No	No	No	No	Yes	No	Yes

L, left; *R*, right; *PU*, proximal ureter; *DU*, distal ureter; *UVJ*, uretovesical junction; *UPJ*, uretopelvic junction; *US*, ultrasound; *CT*, computed tomography; *EDUS*, emergency department ultrasound

Ultrasound is safe, and is now recommended by the European Association of Urology as the first-line imaging modality in suspected renal colic.[13] Until American clinicians move away from CT and begin ordering formal US studies for these patients, emergency physicians will likely continue to perform most of the renal colic ultrasound studies.

Direct sonographic visualization of the ureters is extremely difficult. Hydronephrosis, therefore, is used as a surrogate finding in patients with suspected renal colic. The sensitivity and specificity of bedside ultrasound for the detection of hydronephrosis have been shown to be 87% and 83% respectively.[14] However, hydronephrosis is not always present and mild hydronephrosis is a subtle finding. In addition, a recent study showed that bedside sonography looking for hydronephrosis had only a limited impact on the diagnostic impression of emergency physicians.[15] Traditional EDUS renal colic studies do not include a search for bladder wall abnormalities and the concepts of looking for these findings and examining their diagnostic implications have not been previously discussed in the literature.

A pseudoureterocele is an obscure term defined as "a lesion causing similar [to a ureterocele] lucent-filling defect at the uretovesical junction on IVU (intravenous urogram)."[16] This case series introduces the bladder bulge as a unifying sonographic description of bladder wall abnormalities seen in patients with ureterolithiasis that includes pseudoureteroceles, focal wall thickening, edema, ureteric prolapse and inward wall protrusion, all at the level of the UVJ. There may be some degree of overlap among the findings. Our cases suggest that the mechanism by which the bladder bulge occurs is not entirely explained by stone impaction at the UVJ. There may be additional unilateral detrusor hyperactivity from inflammatory mediators released during ureteral injury. Prostanoid and endothelin subtypes are known to directly contract human detrusor muscle.[17] This would explain the inward wall protrusion seen in cases 5, 6 and 7. All of these patients first underwent EDUS then later had CTs confirming the presence of stones *considerably proximal* to the UVJ. We believe that this particular finding has not been previously described in the literature.

LIMITATIONS

This report is a case series. All cases were identified retrospectively and some had incompletely filled bladders. Potential stone migration during the interval between US and CT makes exact stone location at the time of US difficult to assess. False positives could theoretically occur with partially filled or inadequately interrogated bladders, adjacent bowel or pelvic masses that may compress the bladder, ureteroceles, bladder malignancy, enlarged prostate, altered anatomy from previous surgery, or normal variance.

CONCLUSIONS

The bladder bulge is a novel sonographic term that describes focal bladder wall thickening or inward wall protrusion at the level of the UVJ in patients with ureterolithiasis. It may not be unique to impacted UVJ stones. We hypothesize that the bladder bulge is commonly present in patients with renal colic, and sonographic visualization of the bulge in patients with suspected renal colic will increase the likelihood of a ureteral stone being present. If so, this finding has the potential to curtail unnecessary CT, especially when used in the presence of hydronephrosis. Emergency physicians should begin to look for the bladder bulge sign when renal colic is suspected. Further studies need to be done to examine the sensitivity and specificity of the bladder bulge sign, both alone and with hydronephrosis and hematuria; the duration it remains visible after stone passage; the relationship of the sign to the stone location; and the ease or difficulty with which the bladder bulge is obtained at the bedside.

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Sonographic Consensual Pupillary Reflex

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Patients suffering from severe orbital trauma are at risk for numerous complications, including orbital compartment syndromes. This can result in an afferent pupillary defect, which must be evaluated for on physical examination. Unfortunately, these at-risk patients are often challenging to examine properly due to surrounding edema. Point-of-care ultrasonography can be used as an adjunct to the standard examination in this situation. [West J Emerg Med. 2012;13(6):524]

A 25-year-old male presented to the emergency department after being assaulted outside of a bar by a bare-fisted assailant resulting in significant head trauma and right periorbital swelling and pain. On physical examination, he was unable to open his eye and his ability to execute extraocular movements was uncertain. There was marked periorbital swelling and ecchymosis preventing adequate visualization of the pupil.

Point-of-care ultrasonography was employed–given that a pupillary examination, assessment for entrapment, and evaluation for retinal integrity are fundamental in the evaluation of ocular trauma. A large amount of ultrasound gel was placed over the closed, affected eyelid, and the pupil was then visualized by having the patient look down while a high-resolution linear array ultrasound transducer (Sonosite M-Turbo with ICTx 13-6MHz transducer, Sonosite, Bothell, WA) was directed transversely across the superior-most portion of the orbit and aiming caudally. A light was shone in the unaffected eye to perform a sonographic consensual pupillary reflex assessment, which as shown in the figure, was normal.

The consensual pupillary reflex assesses the integrity of the retina, optic nerve, portion of the midbrain, and the oculomotor nerve in one examination maneuver.[1] Abnormalities of this reflex arc assist in the rapid diagnosis of intracranial hemorrhage, retrobulbar hematoma and other emergent conditions.[2] We describe a novel technique for performing this physical examination maneuver that is useful for patients with orbital edema, injury, or pain that limits proper examination otherwise.

Video. Songraphy of pupillary constriction.

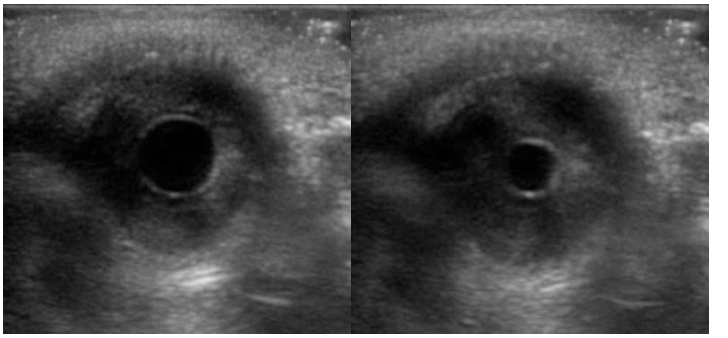

Figure. B-mode view of pupil without light administration (left). B-mode view of pupil during light administration (right).

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Transjugular Intrahepatic Portosystemic Shunt (TIPS) Migration to the Heart Diagnosed by Emergency Department Ultrasound

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A 57-year-old man presented to our emergency department with altered mental status. He had a past medical history significant for cirrhosis and previous placement of a transjugular intrahepatic portosystemic shunt (TIPS). On cardiac auscultation, a new heart murmur and an unexpected degree of cardiac ectopy were noted. On the 12-lead electrocardiogram, the patient was noted to have multiple premature atrial contractions, corroborating the irregular heart rhythm on physical exam. A focused bedside emergency ultrasound of the heart was then performed. This exam revealed an apparent foreign body in the right atrium. It appeared as if the patient's TIPS had migrated from the heart into the right atrium. This case, as well as the literature describing this unusual complication of TIPS placement, is reviewed in this case report. [West J Emerg Med. 2012;13(6):525-526]

INTRODUCTION

Transjugular intrahepatic portosystemic shunt (TIPS) placement is a common procedure performed in patients with complications of portal hypertension due to cirrhosis. This procedure is primarily used in the management of variceal bleeding refractory to endoscopic and medical therapy, as well as in cases of severe ascites. TIPS results in a permanent intrahepatic tract that typically lies between the right hepatic and the right portal vein, shunting blood away from hepatic sinusoids and thereby reducing portal venous pressure.[1] Patients often experience an improvement in their liver disease following the procedure. Unfortunately, the procedure may also result in complications, the most important of which are deterioration in liver function and hepatic encephalopathy. These complications are related to increased shunting of blood away from the liver with reduced sinusoidal flow.[2] Migration of TIPS into the heart is a rare complication, but has been previously described in the literature.[3-4] In this case report presented, we describe a diagnosis of TIPS migration into the heart made in the emergency department (ED) by bedside emergency ultrasound (EUS).

CASE REPORT

A 57-year-old Hispanic male presented with a 1-day history of altered mental status. He had a past medical history significant for alcohol abuse, hepatitis C and Child-Pugh Class B cirrhosis. He had undergone TIPS placement an unknown number of years before presentation to the ED. Additionally, he had been previously hospitalized for hepatic encephalopathy due to noncompliance with his medical regimen.

On physical examination, the patient appeared comfortable and calm. He was alert, but oriented to name only. Vitals signs were temperature 98.1°F pulse 78 beats/min, respiratory rate 16 breaths/min and blood pressure 130/89 mmHg. The patient was noted to have scleral icterus, and his abdominal exam revealed moderate ascites without tenderness, rebound, or guarding. Unexpectedly, on cardiac auscultation, the patient was noted to have a 2/6 systolic and a 2/6 diastolic murmur with ectopy. A 12-lead electrocardiogram (ECG) was obtained in addition to standard laboratory studies to elucidate the cause of the patient's altered mental status.

The serum white blood cell count was 6,500/mm^3 without neutrophilic predominance, hemoglobin of 10 g/dL, BUN of 10 mg/dL and a creatinine of 0.6 mg/dL. The patient was noted to have an elevated ammonia level at138 umol/L. The 12-lead ECG showed normal sinus rhythm with multiple

premature atrial contractions. To further assess cardiac function, a bedside EUS was performed. This exam showed an apparent foreign body in the right atrium (Video 1). The object could be traced back to the liver and was consistent with a TIPS that had migrated distally into the heart. On closer evaluation using color doppler ultrasound, the patient was noted to have an atrial septal defect. The stent appeared to be located close to this defect, although it had not yet crossed the midline (Video 2).

The patient was admitted to the hospital for management of his hepatic encephalopathy, as well as for evaluation of the aberrantly located TIPS. During his hospitalization, the patient underwent a formal right upper quadrant ultrasound, which revealed a patent TIPS without definite portal venous clot. The patient did not have further episodes on ectopy as an inpatient. After review with interventional radiology, his case was deemed amenable to outpatient TIPS evaluation with a catheter snare and replacement procedure.

DISCUSSION

TIPS placement is a common, relatively non-invasive procedure used in patients with complications of portal hypertension. In this case report, a rare complication of this medical device is described. Cardiac complications of this procedure are uncommon, but include arrhythmias during or after placement. Structural damage to the heart by TIPS migration or extension into the cardiac chambers is rare, even though the distance from the hepatic vein-inferior vena cava take-off and the right atrium is only 1.6-2.2 cm.[3] Various cardiac injuries caused by TIPS migration have been previously described, including tricuspid valve injury, atrial-aortic fistula, cardiac rupture with hemopericardium, atrial septal perforation, and heart failure.[3,5-7] The stent may either partially migrate with subsequent damage caused by the cephalad end of the stent or become completely dislodged as a free intracardiac foreign body.[5] Management of this complication most commonly involves percutaneous techniques generally performed by interventional radiologists.[8] Occasionally, open cardiotomy is necessary.[9]

The use of bedside EUS has become standard practice for evaluation of an expanding list of critical diagnoses. The ability of the emergency physician to perform detailed cardiac and abdominal ultrasound provides critical information early in the course of the evaluation of the ill and injured patient. The case reported here illustrates the use of this exam in allowing for the immediate diagnosis of TIPS migration into the heart of a patient with advanced cirrhosis and hepatic encephalopathy. Migration of a TIPS should be considered in any patient with a new heart murmur, signs of right heart strain or dysrhythmia. Focused EUS may then be employed to make the diagnosis of this unusual, but important and potentially life-threatening complication.

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Unusual Cause of Cardiac Compression in a Trauma Patient: Cystic Thymoma

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A 29-year-old man with sudden onset of dyspnea and chest pain with impairment of the general status after falling down from five meters was transferred to our emergency department. He was completely asymptomatic before the injury, but hypotensive (80/50 mmHg) and tachycardic (112 beats/minute) after the injury. Chest radiograph revealed a bulging cardiac silhouette on the right paracardiac region with an increased cardiothoracic ratio of 70% (Figure 1a). Computed tomography revealed a giant cystic mass with a diameter of 10 cm making cardiac compression (Figure 1b). On transthoracic echocardiography, a huge mass neighboring the right atrium with 15x10 cm dimensions was seen. The mass was outside of the pericardial cavity. The left ventricular ejection fraction (LVEF) was 40%. The patient was taken to urgent operation. A huge unruptured cystic mass, which had no connection with the pericardium, was easily resected. Histopathologically, it was reported as a cystic thymoma. On the third postoperative day, echocardiography showed normal cardiac functions with a LVEF of 60%. The patient was discharged without any complications and asymptomatic after three months following surgery.

Thymomas are the most common anterior mediastinal masses in adults, accounting for 15% all mediastinal tumors and 30 to 50% of them are asymptomatic.[1,2] It is reported that 40% of thymomas are characterized by cystic degeneration.[3]

In the present case, the huge cystic thymoma was present in the anterior mediastinum for many years without any symptoms. It was firstly misdiagnosed as pericardial

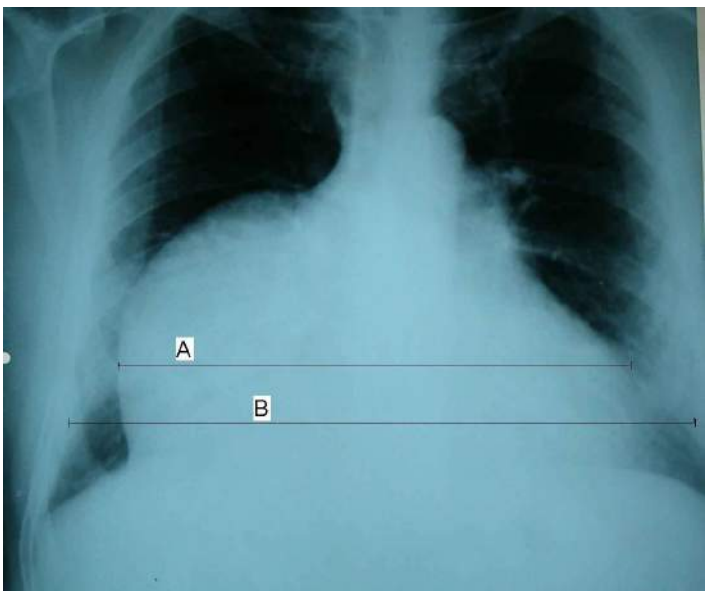

Figure 1a. Posteroanterior chest radiogram showing increased cardiothoracic ratio (brackets). The cardiac diameter (a) is 70% of the transthoracic diameter (b).

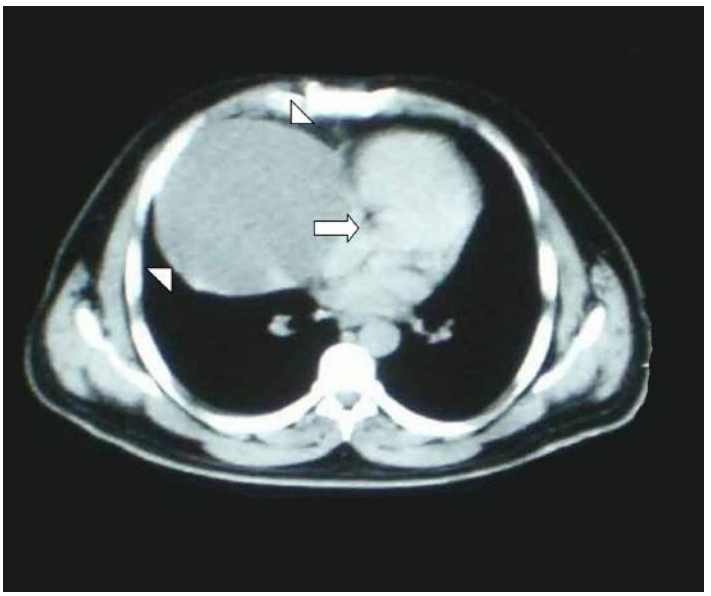

Figure 1b. Contrast-enhanced computed tomography showing a large cystic mass (arrowheads)causing cardiac compression (arrows).

tamponade because of a history of injury. A giant cyst formation compressing the heart became obvious after imaging. Although a definitive diagnosis can be obtained by the histopathological examination of the operative material, cystic thymoma should be considered in patients with anterior mediastinal cystic masses.

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Grade III or Grade IV Hypertensive Retinopathy with Severely Elevated Blood Pressure

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Introduction: Hypertensive retinopathy describes a spectrum of retinal changes in patients with elevated blood pressure (BP). It is unknown why some patients are more likely to develop acute ocular end-organ damage than others with similar BP. We examined risk factors for grade III/IV hypertensive retinopathy among patients with hypertensive urgency in the emergency department (ED) and compared healthcare utilization and mortality between patients with and without grade III/IV hypertensive retinopathy.

Methods: A preplanned subanalysis of patients who presented to a university hospital ED with diastolic BP $\geq$ 120 mmHg and who enrolled in the Fundus Photography versus Ophthalmoscopy Trial Outcomes in the ED study was performed. Bilateral nonmydriatic ocular fundus photographs, vital signs, and demographics were obtained at presentation. Past medical history, laboratory values, healthcare utilization, and mortality were ascertained from medical record review at least 8 months after initial ED visit.

Results: Twenty-one patients with diastolic BP $\geq$ 120 mmHg, 7 of whom (33%) had grade III/IV hypertensive retinopathy, were included. Patients with retinopathy were significantly younger than those without (median 33 vs 50 years, $P = 0.02$). Mean arterial pressure (165 vs 163 mmHg) was essentially equal in the 2 groups. Patients with retinopathy had substantially increased but nonsignificant rates of ED revisit (57% vs 29%, $P = 0.35$) and hospital admission after ED discharge (43% vs 14%, $P = 0.28$). One of the patients with retinopathy died, but none without.

Conclusion: Younger patients may be at higher risk for grade III/IV hypertensive retinopathy among patients with hypertensive urgency. Chronic compensatory mechanisms may have not yet developed in these younger patients. Alternatively, older patients with retinopathy may be underrepresented secondary to increased mortality among these patients at a younger age (survivorship bias). Further research is needed to validate these preliminary findings. [West J Emerg Med. 2012;13(6):529–534.]

INTRODUCTION

Hypertensive retinopathy describes a continuum of retinal changes in patients with elevated blood pressure (BP).[1] While various predisposing factors for malignant hypertension have been suggested and several risk factors for grade I/II hypertensive (ie, chronic, low grade) retinopathy (Figure 1) have been defined, the factors that lead to acute ocular end-organ damage, defined as grade III/IV hypertensive retinopathy

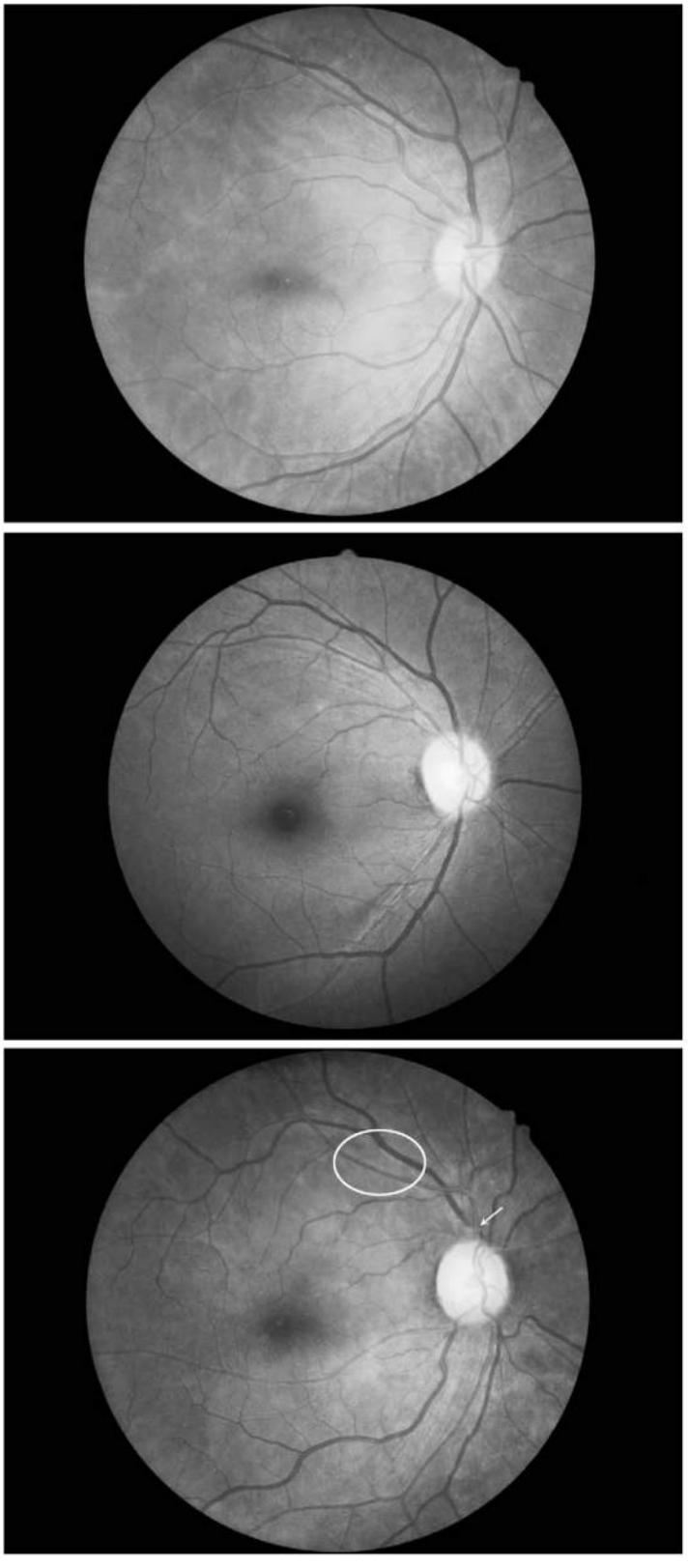

Figure 1. Normal versus grade I/II hypertensive retinopathy. (Top) Normal ocular fundus. Note that the ratio between the arteries and the veins (arteriovenous [AV] ratio) is about 2:3. (Middle) Grade I hypertensive retinopathy. Note mild narrowing and sclerosis of retinal arteries with an overall AV ratio of about 1:2. (Bottom) Grade II hypertensive retinopathy. There is AV nicking (arrow) and moderate to severe narrowing and sclerosis of arterioles (eg, within the ellipse the AV ratio is less than 1:2).

(Figure 2), are less well characterized.[2–10] Additionally, little is known regarding the correlation between high-grade hypertensive retinopathy and the use of healthcare resources. Determining the risk factors for grade III/IV hypertensive retinopathy and the condition's effects on future healthcare utilization may guide examination and treatment strategies in the emergency department (ED) setting.

We undertook a pilot examination of risk factors for grade III/IV hypertensive retinopathy based on our hypothesis that chronic hypertensive changes in the ocular fundus may be indicative of protective mechanisms against acute end-organ damage, exhibited as grade III/IV hypertensive retinopathy. We aimed to define patient characteristics that may predispose to the development of grade III/IV hypertensive retinopathy and to relate the presence of hypertensive retinopathy to mortality and healthcare utilization.

METHODS

Our institutional review board approved the study. Between March 2009 and January 2010, 350 subjects were enrolled in the Fundus Photography versus Ophthalmoscopy Trial Outcomes in the Emergency Department (FOTO-ED) study using a brief, modified written informed consent. Detailed methods of the FOTO-ED study are described elsewhere.[11,12] Briefly, the FOTO-ED study was an evaluation of adult patients who presented to an academic, tertiary care hospital ED with a chief complaint of headache, acute focal neurological dysfunction, or acute visual changes; or who had a triage diastolic blood pressure (DBP) $\geq$ 120 mmHg. Patients were excluded if they were unable or unwilling to sit up, were not interested in participating, were confused, or were otherwise unable to consent. In the present study, only the subset of patients with DBP $\geq$ 120 mmHg were evaluated as part of a preplanned subanalysis.

Undilated fundus photographs of both eyes were taken by trained nurse practitioners using the Kowa nonmyd-α-D fundus camera (Torrance, California). Photographs were reviewed by 2 neuro-ophthalmolgists for the presence or absence of grade III/IV hypertensive retinopathy, according to the Keith, Wagener, and Barker classification (Table 1) by consensus (a third ophthalmologist was available for arbitration, but not needed).[13,14]

Demographics; height; weight; all vital sign measurements; prior diagnoses of diabetes, hypertension, left ventricular hypertrophy, and congestive heart failure; smoking history; pertinent lab values; and treatment regimens were determined from review of medical records. Body mass index (BMI) was calculated as weight (kg)/height $(m)^2$. Mean arterial pressure (MAP) was calculated, according to the method of

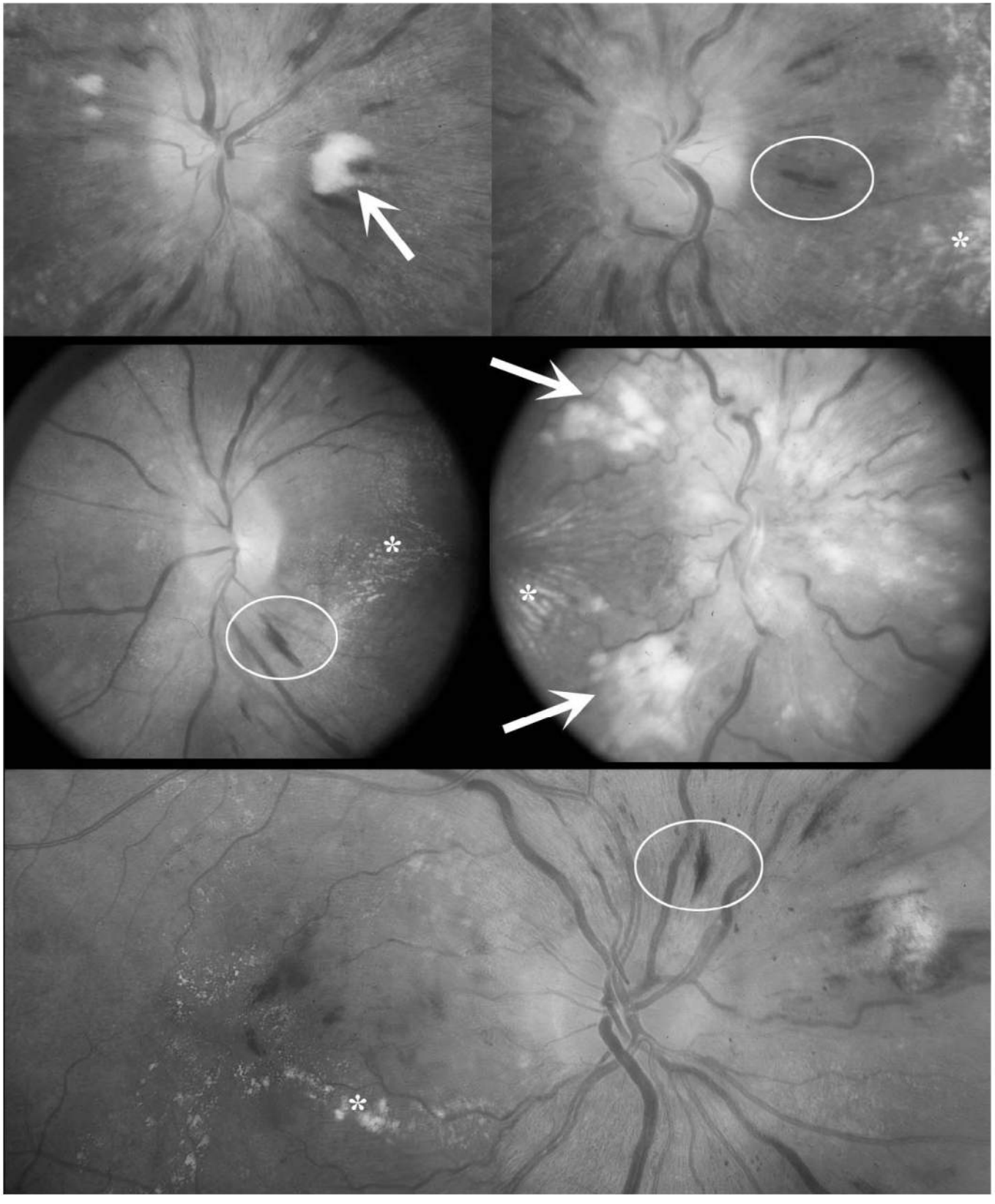

Figure 2. Examples of grade III/IV hypertensive retinopathy. Note the features of grade III retinopathy: exudates (asterisks), cotton wool spots (arrows), and nerve fiber layer hemorrhages (ellipses). Grade IV hypertensive retinopathy is defined by the presence of features of grade III retinopathy plus optic nerve head edema (eg, the middle right panel).

Table 1. Keith, Wagener, and Barker classification of hypertensive retinopathy.[13,14]

Grade I: Slight or modest narrowing of the retinal arterioles, with an arterial:venous ratio of ≥1:2.
Grade II: Modest to severe narrowing of retinal arterioles with an arterial:venous ratio <1:2 or arteriovenous nicking.
Grade III: Soft exudates and flame-shaped hemorrhages.
Grade IV: Grade III changes and bilateral optic nerve edema.

Razminia et al, as DBP + (0.33 + [0.0012 × HR]) × (PP), where HR = heart rate and PP = pulse pressure.[15]

Retinal arteriovenous ratio (AVR) and retinal arteriolar tortuosity measures were determined from fundus photographs using ImageJ (NIH, Washington, DC). The AVR was calculated, incorporating aspects of the method of Parr and Spears.[16,17] Tortuosity was determined based on the method of Lotmar et al.[18] See the Appendix (online only) for further details of AVR and tortuosity measurements.

Medical records were also examined to ascertain the number and dates of ED visits and hospital admissions. As many as 16 months and no fewer than 8 months of follow-up from enrollment in the study were available. Neither the initial ED visit nor a hospital admission occurring at that time was included in this data. The Social Security Death Index and hospital records were reviewed to determine mortality during this time period.

Statistical analysis was performed using R: a language and environment for statistical computing (R Foundation for Statistical Computing, http://www.r-project.org). We compared the characteristics of individuals with DBP ≥ 120 who had grade III/IV hypertensive retinopathy to those without grade III/IV hypertensive retinopathy. We performed univariate analyses on each of the variables to produce summary measures for grade III/IV hypertensive retinopathy as the outcome for each potential risk factor, using the Wilcoxon rank sum test for continuous variables and the Fisher exact test for discrete variables. Here, P values < 0.05 were considered statistically significant. In cases of missing data, the number of subjects included in each analysis was reported. The Bartlett test was used to compare the variance in systolic BP (SBP) and DBP between the 2 groups. Summary measures stratified by age were also calculated.

RESULTS

We identified 21 subjects with DBP ≥ 120 mmHg among 350 subjects enrolled in the FOTO-ED study. Of these, 7 (33%) had grade III/IV hypertensive retinopathy on examination of ocular fundus photographs (Table 2). Here, MAP (median 165 vs 163, $P = 0.80$), SBP (median 212 vs 212, $P = 0.88$), and DBP (median 123 vs 125, $P = 0.62$) were essentially the same in both groups. Subjects with grade III/IV hypertensive retinopathy were significantly younger than those without retinopathy (median age 34 vs 50 years, $P = 0.02$). The youngest age quartile (19–36 years) had a 67% risk of hypertensive retinopathy, double that of the second quartile (36–46 years: 33%), and more than triple that of the third quartile (46–62 years: 20%). There were no cases of grade III/IV hypertensive retinopathy in the fourth quartile (62–75 years). There was no difference in retinal arteriolar tortuosity (0.064 vs 0.08, $P = 0.62$) or AVR (0.81 vs 0.78, $P = 0.48$) between the 2 groups. There were no significant differences in sex, race, BP, BP variation, heart rate, BMI, or random serum glucose measurements between the 2 groups. Subjects in the 2 groups were similar in terms of past medical history, including all secondary measures examined. All subjects had a history of diagnosed hypertension, and antihypertensive treatment rates were not significantly different between the 2 groups with (100%, n = 7) and without (77%, n = 13, 1 patient could not recall his medications) grade III/IV hypertensive retinopathy ($P = 1.00$).

The patterns of healthcare utilization between the 2 groups were not significantly different, but there was a 2-fold higher proportion of patients who returned to the ED (57% vs 29%, $P = 0.35$) and a 3-fold higher proportion of patients who were admitted to the hospital at a later date (43% vs 14%, $P = 0.28$), when patients with grade III/IV hypertensive retinopathy were compared to those without. One subject with grade III/IV hypertensive retinopathy died during the follow-up period. There were no deaths in the group without grade III/IV hypertensive retinopathy.

DISCUSSION

This study found that younger age was a strong risk factor for the development of grade III/IV hypertensive retinopathy independent of BP. Indeed, we found that the median age of patients with grade III/IV hypertensive retinopathy was 16 years younger than those without. Furthermore, the relationship between younger age and the development of grade III/IV hypertensive retinopathy is consistent with our a priori hypothesis that chronic hypertension may have effects protective against the development of acute hypertensive damage. To our knowledge, there are no studies that examine the effect of chronic hypertensive change on the development of acute retinal hypertensive change. While our study did not show evidence that chronic hypertensive ocular changes (eg, smaller AVR) were protective, this may have been due to our lack of power to detect such a change in our small sample.

Age as a risk factor for grade III/IV hypertensive retinopathy could alternatively be explained by a survivorship bias; ie, individuals at risk for developing grade III/IV hypertensive retinopathy may have higher mortality rates, thus leaving fewer of these patients in the older age groups of a cross-sectional study. Consistent with this theory, our patients with grade III/IV hypertensive retinopathy demonstrated an increased likelihood for return to the ED at a later date, hospital

Table 2. Characteristics of patients with hypertensive urgency and grade III/IV hypertensive retinopathy compared with the remainder of the study population with hypertensive urgency.

	Grade III/IV hypertensive retinopathy (n = 7)		No grade III/IV hypertensive retinopathy (n = 14)		
	Number or median	% or range	Number or median	% or range	*P*
Demographics/risk factors					
Women	3	43%	10	71%	0.35
Age (years)	34	21 to 62	50	19 to 75	0.02*
White	1	14%	3	21%	1.00
Systolic blood pressure	212	173 to 259	212	185 to 242	0.88
Diastolic blood pressure	123	120 to 131	125	120 to 143	0.62
Mean arterial pressure	165	143 to 178	163	149 to 184	0.80
Heart rate	86	60 to 101	85	65 to 110	0.88
Body mass index	25	21 to 34	32	25 to 53	0.16
Medical history					
Diabetes mellitus	2	29%	1	7%	0.25
Left ventricular hypertrophy†	5	71%	4	31%	0.21
Congestive heart failure	1	14%	1	7%	1.00
Smoking†	4	57%	6	46%	1.00
Treatment: Home BP medications prescribed†	7	100%	10	77%	1.00
Lab: Random serum glucose†	94	74 to 187	104	77 to 115	0.22
Retinal vessel signs					
Average AVR‡	0.81	0.72 to 0.87	0.78	0.51 to 0.91	0.48
Average tortuosity§	0.064	0.03 to 0.52	0.08	0.02 to 0.24	0.62
Healthcare utilization					
≥1 ED	4	57%	4	29%	0.35
≥1 hospital admission	3	43%	2	14%	0.28

AVR, arterial:venous ratio; *BP*, blood pressure; *ED*, emergency department.
* Statistically significant.
† One patient without grade III/IV hypertensive retinopathy without data.
‡ One patient with and one without grade III/IV hypertensive retinopathy had fundus photos ungradable for AVR.
§ One patient with grade III/IV hypertensive retinopathy had fundus photos ungradable for tortuosity.

admission at a later date, and death (ED revisit: 57% vs 29%, hospital admission: 43% vs 14%, death: 14% vs 0%), and low statistical power in our study may account for this the lack of statistical significance, especially given consistent direction of these effects.

LIMITATIONS

The main limitations of our study are the small sample size and modest follow up after patients were discharged. The small sample size resulted in a low power to detect significant differences between study groups. However, this study was intended to be a pilot investigation with the purpose of identifying possible risk factors and generating hypotheses for further investigation. Additionally, follow up of ED visits and hospital admissions were restricted to healthcare that took place within our own institution. Because participants were not directly contacted after their ED visit, it is possible that some patients returned to an outside ED or hospital. This occurrence could have introduced bias into our results, but only if patient visits to outside healthcare facilities differed between the 2 groups. Inclusion of larger patient samples and continued contact with study participants after discharge from the ED would enhance future studies on this topic.

CONCLUSION

In conclusion, our pilot study suggests that grade III/IV hypertensive retinopathy may increase future healthcare utilization, in terms of ED visits and hospital admissions, and possibly mortality, although these results did not reach statistical significance. Most concerning, patients in our study with grade III/IV hypertensive retinopathy were more likely to be young. Emergency physicians should be aware that younger

patients are at a higher risk for ocular end-organ damage and should ensure that all patients with severely elevated BP receive an appropriate screening examination of the ocular fundus for signs of acute end-organ damage. This is particularly important because the presence or absence of severe, grade III/IV hypertensive retinopathy in patients with BP exceeding 180/120 mmHg is one feature that helps differentiate less severe hypertensive urgencies from hypertensive emergencies, which require intensive care according to the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7).[19]

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"Challenging the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage, and Shaken Baby Syndrome"

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Gabaeff SC. Challenging the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage, and Shaken Baby Syndrome. West J Emerg Med. 2011;12(2):144-158.

To the Editor:

As an ophthalmologist, I was very interested in the article by Gabaeff and the response by Greeley.[1,2] Greeley is familiar with the contributions Vinchon has made in this area, even referencing one of his articles. Since this response was written well after Vinchon's paper concerning "spontaneous" intracranial and intraocular hemorrhage, this reference would appear highly relevant.[3] For example, Vinchon points out the importance of increased cerebral spinal fluid spaces. Greeley correctly notes the case from Rooks had such spaces but then says the child did not have hydrocephalus, when in fact extra-axial fluid collections are just one of many names given to this condition, benign external hydrocephalus (BEH) being another. Greeley "having board certification in both general pediatrics and child abuse pediatrics, and having experience and training in clinical research and medical literature appraisal" is certainly aware of this. He also has previously discussed this very problem in a response to one of my articles.[4] Greeley appears unaware of the importance of this when he says Gabaeff's comment concerning non-abuse reasons for this combination is "not supported by the medical literature." He also accuses Gabaeff of not citing anything when talking about the American Academy of Ophthalmology's role in this area. In fact Gabaeff says "as discussed above,"a very definite self citation. If Greeley does not believe Gabaeff's previous statements were adequately supported, this is an entirely different matter. In light of Piatt's previous paper and Vinchon's recent verification of this problem, Gabaeff's concerns that chronic SDH in infants may be being misdiagnosed as abuse seems appropriate.[5]

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Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The author disclose that he has given trial testimony and consulted concerning eye findings in suspected cases of child abuse and shaken baby syndrome approximately 6 times per year for the past 8 years.

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The psychopharmacology of agitation: consensus statement of the American association for emergency psychiatry project BETA psychopharmacology workgroup.

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Wilson MP, Pepper D, Currier GW, Holloman GH, Feifel D. The psychopharmacology of agitation: consensus statement of the American association for emergency psychiatry project BETA psychopharmacology workgroup. *West J Emerg Med.* 2012; 13:26-34.

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To the Editor:

We were excited to read the article by Michael Wilson et al[1] in the March 2012 issue of the *Western Journal of Emergency Medicine* regarding pharmacologic strategies for the management of agitated patients in the emergency setting. This article highlights several important points including the optimal management of stimulant-induced agitation and the feasibility of and reasons for differentiating acute alcohol intoxication from withdrawal, as optimal pharmacologic interventions for each might vary.

While the authors correctly highlight the importance of preferential use of benzodiazepines to calm patients intoxicated with most recreational drugs, we believe that the use of benzodiazepines as first line treatment for agitation should be extended to include that from acute overdose of other agents.

Because many common medications taken in acute overdose, such as cyclic antidepressants, SSRIs, diphenhydramine and other over-the-counter medications have toxicity profiles that include anticholinergic, proconvulsant, hyperthermic, and cardiotoxic (QTc prolongation) properties, which overlap with antipsychotics, we recommend benzodiazepines as first, second and third line for agitation in these instances to avoid contributing to these potentially life threatening adverse effects. Further, benzodiazepines raise the seizure threshold and promote conditions that precipitate heat dissipation.[2,3] If, after liberal use of benzodiazepines, the patient still displays agitation necessitating further pharmacologic intervention, we then use antipsychotics with caution. We found the reported maximum daily dose of lorazepam in the Table[1] to be dangerously low.

We were glad to see the emphasis on patient and staff safety, given the increasing awareness of the excited delirium syndrome, thought to be due to a multifactorial interaction of delirium and agitation often secondary to stimulant intoxication, leading to hyperthermia, profound acidemia and sometimes death.[4-7] We regret that the authors left out a discussion of the increasing use of the dissociative agent ketamine for rapid control of dangerous behavior in this subset of patients. Although no controlled trials exist regarding its use in agitated patients, several case reports show rapid, satisfactory results without significant respiratory and cardiovascular adverse effects.[5,8] Potential adverse effects of ketamine, although uncommon, include hypertension, emergence phenomena, increased oral secretions and laryngospasm.[7,10]

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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In reply:

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We appreciate the thoughtful comments by Drs. Hays, Jolliff and Casavant regarding the consensus guidelines we authored for the pharmacologic management of agitated patients in

the emergency setting.[1] They disagree with the fact that these guidelines do not recommend benzodiazepines as first line treatments in all cases of agitation associated with substance intoxication because many compounds taken in acute overdose have a propensity to produce anticholinergic, proconvulsant, hyperthermic, and cardiotoxic (QTc prolongation) effects which overlap with antipsychotics but not benzodiazepines.

Our guidelines divide agitation secondary to intoxication into that which is primarily caused by CNS stimulants and that which is caused primarily by CNS depressants, most notably alcohol. Benzodiazepines are recommended as first line in the guidelines for the former category, while an antipsychotic drug (preferably non-sedating) is recommended for the latter. While we recognize patients displaying agitation in an emergency setting often have more than one substance on board, we believe the division of intoxication-induced agitation into these two categories present clinicians with a conceptual road map for decision making. The overtly alcohol-inebriated, agitated patient is the representative patient we had in mind for the CNS depressant category. Acute alcohol ingestion is not strongly associated with any of the physiological effects that Drs. Hays, Jolliff and Casavant cite. On the other hand, both benzodiazepines and alcohol share a propensity toward respiratory depression and combined they pose an additive or even synergistic potential risk of respiratory depression.[2-4] On this basis we did not recommend benzodiazepines as first line treatment for agitation in a patient whose presentation is highly consistent with alcohol as the primary intoxicant.

We would also like to point out a common misperception, alluded to in the letter by Drs. Hays, Jolliff and Casavant, that antipsychotics produce hyperthermia. While in certain rare situations, excessively high doses of (mostly first generation) antipsychotics can produce NMS, a syndrome associated with hyperthermia, under normal circumstances antipsychotics tend to lower body temperature.[5]

Additionally, we share the interest, expressed by Drs. Hays, Jolliff and Casavant, in ketamine as a potential agent in the treatment of patients described as having "Excited Delirium Syndrome." However, as they note in their letter, despite growing clinical experience and several case reports supporting its use in this putative, specific subgroup of agitated patients, there is, as of yet, a dearth of high quality evidence (i.e. controlled trials) regarding the safety and efficacy of this treatment relative to other established treatments for agitation. There is also no reliable method, as of yet, for identifying patients who may be well suited for ketamine and those for whom it may be contraindicated. For example, patients with untreated psychotic disorders, such as schizophrenia, are considered to represent a substantial portion of the patients who present with "excited delirium."[6] The psychotomimetic nature of ketamine raises the distinct possibility that it may exacerbate the underlying psychosis in these patients. Moreover, recreational ingestion of ketamine and PCP, which is a derivative of ketamine and shares its antagonism of NMDA subtype glutamate receptors, are known to induce an "excited delirium" presentation.[7] In a patient whose agitation is due to ketamine or PCP, administration of ketamine would exacerbate the underlying pharmacological toxicity. For these reasons we felt that clinical knowledge regarding excited delirium syndrome and the use of ketamine in these situations has not, at this time, sufficiently matured to include it among recommended treatments.

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Holiday Plants with Toxic Misconceptions

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Several plants are used for their decorative effect during winter holidays. This review explores the toxic reputation and proposed management for exposures to several of those, namely poinsettia (*Euphorbia pulcherrima*), English holly (*Ilex aquifolium*), American holly (*Ilex opaca*), bittersweet (*Solanum dulcamara*), Jerusalem cherry (*Solanum pseudocapsicum*), American mistletoe (*Phoradendron serotinum*), and European mistletoe (*Viscum album*). [West J Emerg Med. 2012;13(6):538-542]

INTRODUCTION

December is a much-decorated month with many holidays, celebrations, and adornments. These very decorations, however, are allegedly toxic. We review the toxic (or not so toxic, in some cases) characteristics of 7 common holiday plants – poinsettia, English and American holly, bittersweet, Jerusalem cherry, and American and European mistletoe.

PLANTS

Poinsettia (*Euphorbia pulcherrima*)

This flowering plant, indigenous to Mexico and Central America, has large green and red leaves. It was reportedly introduced to the United States in 1829 by J.R. Poinsett, the American ambassador to Mexico at the time.[2] In the wild it is a large, woody shrub commonly growing 10 feet high. Indoors it is typically much smaller with denser leaves.[3] It is commonly used as a Christmas decoration; as a result, the majority (74.9%) of exposures are in the months of December, January, and February.[4] The most recent annual report of the American Association of Poison Control Centers' National Poison Data System (NPDS) revealed that *Euphorbia* had the 6th highest rate of genus-specific human plant exposure calls in 2010.[5] The number of poinsettia-related calls for the year was 750, which likely underestimates the actual amount of exposures.[5]

Despite a long-standing belief in the legend of poinsettia toxicity, there is little data to support this. The toxic reputation stems from a single unconfirmed death of a 2-year-old in Hawaii in 1919. While the *Euphorbia* genus contains complex terpenes (diterpenes) that are local irritants and cause gastrointestinal upset, the *pulcherrima* species does not contain this toxin.[6,7]

Figure 1. Poinsettia (*Euphorbia pulcherrima*)[1]

Poinsettia is in the same plant family as natural rubber latex and shares 2 common allergen proteins. Forty percent of individuals with a latex allergy develop cross-sensitivity with the poinsettia plant.[8] Symptoms vary from rare immediate hypersensitivity (type I), allergic contact dermatitis (type IV), or irritant contact dermatitis. Case reports of anaphylactic shock from poinsettia in infants with atopic eczema and latex allergies have been published with probable causality, although these are a rarity. Families that include members with atopic eczema or other generalized atopia may want to avoid using poinsettia as a decorative addition to their holiday.[9]

A vast majority of exposures, however, result in either no effect or minor gastrointestinal upset and nausea with

Table. Holiday plants with respective toxin and clinical effects.

Plant	Toxin	Clinical Effects
Poinsettia (*Euphorbia pulcherrima*)	None identified	Minor gastrointestinal upset/cramping, dermatitis
Holly (*Ilex aquifolium* and *opaca*)	Saponin	Gastrointestinal upset/cramping, dermatitis Uncommon: mydriasis, hyperthermia, ataxia, muscle weakness, dyspnea, drowsiness, altered mental status
Bittersweet (*Solanum dulcamara*), Jerusalem Cherry (*Solanum pseudocapsicum*)	Solanine	Gastrointestinal upset/cramping Uncommon: salivation, bradycardia, hypotension, altered mental status
	Dulcamarine	Anticholinergic symptoms
Mistletoe (*Phoradendron serotinum* and *Viscum album*)	Toxalbumins	Gastrointestinal upset/cramping with necrotic sloughing of gastrointestinal tract Uncommon: bradycardia, delirium, and liver/central nervous system/renal/adrenal toxicity

occasional vomiting.[7] Contact dermatitis is very rare. Krenzelok[4] reviewed 22,793 cases of poinsettia exposure that were collected by poison control centers (PCCs) from 1985-1992. Of these, 98.9% were accidental and no fatalities were observed. The vast majority of exposures were ingestions (94.5%), with some dermal exposures as well (4.8%). In part due to the attractiveness of the poinsettia's foliage, nearly all of ingestions are in children (93.3%), with the majority in children younger than 2 years (77.3%). Ninety-six percent were not referred by their respective PCC to a healthcare facility. In addition, 92.4% did not develop any toxicity, and 3.4% only had minor clinical effects.[4] The clinical effect seen with 1 particularly large ingestion was minor gastrointestinal upset and abdominal cramping.

The overriding results of multiple attempts to analyze potential toxicity using animal models revealed little to no toxicity. One study could not find a lethal dose 50 (dose at which 50% of the exposed subjects expire) in rats. At the highest dose orally administered (25 g/kg) there was no evidence of any symptoms during a 14-day observation period or any toxicity noted on subsequent autopsy. The same study could find local irritation/inflammation on repeated instillation into the buccal cavity of rats and the eyes of rabbits. There was some minor skin irritation with repeated exposure in rabbits.[10] These findings are echoed in other studies.[11,12]

Nearly all patients do not require any therapy and can be treated without healthcare facility referral. In those that do present to an emergency department (ED), the induction of emesis, decontamination, and the use of dilution appear to be of little or no value and are not recommended.[4] Supportive care through symptomatic management, such as antiemetics, should be all that is necessary.

Holly (*Ilex aquifolium* and *opaca*)

There are 2 commonly distributed forms of the holly in the United States (U.S.): the English holly (*Ilex aquifolium*) and the American Holly *(Ilex opaca*). Holly is a small tree or shrub that will grow up to 15 m tall and carries scarlet-red berries approximately 10 mm in diameter.[3] English and American holly are not to be confused with the South American *Ilex* species, *Ilex paraguariensi* and *Ilex guayusa*, which are commonly used to make teas and other drinks for their reported antioxidant properties and caffeine content.[14,15] These shrubs are most commonly used as holiday decorations, although they can be found in gardens. Holly exposure accounts for the 3rd highest rate of genus-specific human plant exposure calls in 2010, with 877.[5] The berries containing the toxin saponin are poisonous; the leaves are not.[7] The toxic component of the berries is saponin.[7,16] The primary potential biological effect of saponin is a negative interaction with cellular membranes. Saponins can cause hemolysis in erythrocytes and alterations in permeability of small intestinal mucosal cells. Most ingestions cause little or no toxicity. The primary clinical effects observed, which occur exclusively with large ingestions, include nausea, vomiting, abdominal cramping, and occasionally dermatitis. There can be allergic sensitization and worsening dermatitis with repeat exposures.[7] Rarely, mydriasis, hyperthermia, and drowsiness have also been reported.[17,18]

Figure 2. Holly (*Ilex aquifolium* and *opaca*)

Rodrigues et al[19] describes a case of 2 identical twins that

ingested a "handful" of holly berries. One twin vomited 40 times over 6 hours and was drowsy, while the other twin had only 5 episodes of emesis in the same time period without drowsiness. Poisonings most often occur in children, and most cases are harmless. In adults, 1 must eat 20-30 berries before becoming symptomatic, whereas children only have to consume 5.[18]

One study attempted to explore management techniques for pediatric ingestions of toxic berries (including holly berries), comparing home observation alone with syrup of ipecac and home observation. Predictably, all of the patients in the ipecac group vomited, while there was no vomiting among the subjects in the home observation alone group. There was more sedation and diarrhea in the ipecac group as well.[20] Ipecac is no longer recommended for toxic ingestions in general. ED therapy recommendations for holly berry exposures include symptomatic management, such as antiemetics, along with fluid and electrolyte supplementation for dehydration from rare severe vomiting and diarrhea.[7,18]

Bittersweet (*Solanum dulcamara*) and Jerusalem cherry (*Solanum pseudocapsicum*)

Bittersweet, or the woody nightshade, is a semi-woody perennial vine introduced from Europe. Common to the northern U.S. and southern Canada,[3] it has purple and yellow flowers with 5 spreading petals and red ovoid berries. The Jerusalem cherry, or Christmas orange, is a perennial grown as a decorative houseplant. Originating in the Middle East, it now flourishes in Hawaii and the Gulf Coast states. It also has 5-petaled flowers but typically has yellow-red-orange berries.[7] *Solanum*-related poison center calls in general are common, and *S. dulcamara* alone made up the 22nd most species-specific U.S. poison center calls, with 406 total.

In both of these plants the immature fruit is more poisonous than the still-toxic ripened fruit due to the glycoalkaloid solanine.[3] Solanine may exert toxicity through alteration of mitochondrial potassium and calcium transport, but this mechanism is speculative.[6] In animals solanine exhibits cholinesterase activity and cardiac glycoside effects, but these effects are not seen in human poisoning.[16]

The clinical effects of solanine are primarily gastroenteritis and abdominal cramping. Salivation, bradycardia, tachycardia, hypotension, and altered mental status have also been documented.[17] Symptoms usually occur several hours after ingestion and may persist for several days. The solanine effects seem more potent in children; in adults, solanine has little toxicity. While 1 source reported that just several bittersweet or Jerusalem cherry berries can prove fatal in children, there has been a single authenticated case of death following ingestion of the berries from bittersweet in a 9-year-old female.[2,3,23] That particular child suffered abdominal pain and vomiting 2-3 days post-ingestion, and subsequently developed pallor with dry skin, hypothermia, tachycardia, but neither delirium nor paralysis. Post-mortem exams revealed

Figure 3. Bittersweet (*Solanum dulcamara*) (left)[21] and Jerusalem cherry *(Solanum pseudocapsicum)* (right)[22]

lesions in the gastrointestinal tract. There have been repeated case series that reported essentially mild solanine symptoms of abdominal distress and other more vague symptoms. The largest case series recorded spanned 319 ingestions; 295 were under the age of 10, and only 9 had likely solanine-related symptoms; none had to be hospitalized.[2] There have been a couple of other notable reports of pediatric *S. dulcamara* ingestions. A 7-year-old creating "make up" from crushed leaves and berries subsequently experienced gastritis, tachycardia, and mydriasis.[24]

Case reports document the rare anticholinergic effects of Solanum, likely due to dulcamarine, an atropine-like compound.[3] A 4-year-old girl had an anticholinergic syndrome treated successfully with physostigmine after she was found playing around a bittersweet plant. Causality in that case was questionable.[25]Additionally there are minimally active alkaloids in these plants -- solasodine (in the flowers) and beta-solamarine (in the roots).[26]

Of the plants discussed thus far, it seems as though bittersweet and Jerusalem cherry constitute the most danger. There is little to no data to support serious toxicity to adults, but there may be some real danger to children. Historically, induced emesis was recommended for *Solanum* ingestion, especially in children, but that doesn't coincide with current decontamination recommendations.[16] Prolonged observation may be necessary for children in the context of high likelihood of ingestion. Management includes intravenous fluids and antiemetics for nausea, vomiting, and dehydration.[7] Phsyostigmine could be considered for obvious anticholinergic presentation.

Mistletoe (*Phoradendron serotinum* and *Viscum album*)

There are 2 plants with the common name "mistletoe" – the American mistletoe (*P. serotinum*) and European mistletoe (*V. album*). Mistletoe is a parasitic perennial with white or translucent berries that can be quite sticky. They grow on the trunks and branches of deciduous trees. *P. serotinum* is widespread in the U.S., hence the American mistletoe moniker. *V. album*, endemic to much of Europe, can

Figure 4. Mistletoe (*Phoradendron serotinum and Viscum album*)[27]

occasionally now be found in the U.S., mainly in California.[7] These plants are common adornments and holiday symbols most commonly sold around Christmas time.

All sections of *P. serotinum* are potentially harmful as they contain phoratoxin, a toxalbumin. Most ingestions result in little physical reaction, although some patients may experience gastrointestinal symptoms. The entire *Viscum spp.* plant is toxic, with the exception of the berries, which are harmless except in very large amounts.[7] Viscotoxins, similar in structure and effect to the phoratoxin toxalbumins found in *Phoradendron spp*, inhibit cellular synthesis, thereby affecting cells with rapid turnover like gastrointestinal mucosa. Viscum (also called viscumin), from *V. album*, can also cause erythrocyte agglutination in vitro; it is unclear if this also occurs in vivo.[6] After a latent period of several hours, clinical effects from viscotoxins can develop and are primarily gastrointestinal upset with potential necrotic lesions resulting in sloughing of the gastrointestinal tract. Bradycardia, delirium, as well as toxicity of the liver, central neverous system, kidney, and adrenals can also occur, although the incidence is not known.[16,17]

Although *Phoradendron* and *Viscum spp* both contain the common name "mistletoe," the *Phoradendron spp* has a relatively lower toxicity compared to *Viscum spp*. The only cases involving deaths likely related to *P. serotinum* are cases in which there were teas infused with the fruits and other parts of the plants.[2] The literature does include reports of fatalities due to *V. album* ingestion, primarily due to its alleged medicinal qualities resulting in excessive use in herbal/therapeutic teas.[29] The only 2 reported deaths from ingestion of mistletoe in the past 25 years were patients who ingested brewed teas.[30]

A case review of 14 patients with American mistletoe leaf or berry ingestions failed to show any symptoms of toxicity. Based on that series, ingestion of 1-3 mistletoe berries or 1-2 leaves is unlikely to produce serious toxicity.[30] Krenzelok et al[31] compiled the largest case review of mistletoe exposure to date, 1,754 exposures reported to PCCs from 1985-1992. Accidental pediatric exposures accounted for the majority of cases (94.7% and 92.1% of cases, respectively). Ninety-five percent of cases were due to ingestion rather than exposure via another route. Of the 72 intentional exposures, 11.1% were suicide attempts. Overall, patient outcomes were excellent with no fatalities and 99.2% having no morbidity. Outcomes were not influenced by gastrointestinal decontamination, as 96.2% of treated patients versus 96.3% of untreated patients remained asymptomatic. Seasonal clustering was also seen with 87% of cases seen November through January. The relatively harmless nature of ingestions of *P. serotinum*, at least in small amounts, was shown in previous work as well.[29]

ED management should be directed toward supportive care for dehydration and vomiting if severe gastroenteritis develops.[7] Based on reported onset of symptoms, an observation period of 6 hours would be reasonable in asymptomatic patients. According to case reviews by both Krenzelok et al[31] and Spiller et al[32], gastrointestinal decontamination was not deemed to influence patient outcome and is not recommended.

CONCLUSION

Concentrations of the toxic agent vary depending on the portion of the plant ingested. For plant ingestions the amount of toxin ingested is usually unknown. While nearly all ingestions are in children, nearly all are also asymptomatic given the low concentrations of toxin in the plants reviewed. However, steeping the plant in hot water ("herbal tea") may result in large amounts of ingested toxin.[17] Serum toxin concentrations are not easily available and are not necessary. Because the vast majority of these plant exposures are asymptomatic, home observation with expectant, conservative management is appropriate. However, if severe gastroenteritis does result supportive care with fluid resuscitation and evaluation of electrolytes, glucose, BUN, creatinine, and urinalysis may be useful.[17]

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Adie's Tonic Pupil

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A 33-year-old woman with no past medical history presented to the emergency department with asymmetric pupils. At 7:30AM while putting on makeup, she noted her pupils were equal in size. One hour later, she developed light sensitivity in her right eye, and soon after noticed her right pupil was significantly enlarged. She denied headache, facial or extremity weakness, dysarthria, or ataxia. On exam, her left pupil was reactive from 4 to 3 mm and her right pupil was sluggishly reactive and 8 mm (Figure). No abnormalities in her visual acuity, extraocular movement or fundoscopic exam were detected. Neurologic consultation was obtained, but the patient had an unremarkable brain computed tomography (CT)/CT-Angiography and magnetic resonance imaging.

A tonic pupil results from parasympathetic denervation at the level of the ciliary ganglion. It is characterized by a large, regular pupil with decreased response to light but preserved or enhanced constriction to accommodation, segmental iris constriction, vermiform movements of the pupillary border, and hypersensitivity to pharmacologic constricting agents.[1-4] The diagnosis was established in consultation with ophthalmology and confirmed with rapid miotic response of the affected pupil to 0.125% pilocarpine drop.[1,2] Most cases are idiopathic, occurring in women 20-40 years of age, and referred to as the Adie's tonic pupil, though this disorder can be due to local disorders within the orbit, including tumor, inflammation, trauma, surgery, ischemia or infection.[3] Most patients do not require any treatment and can be reassured once the diagnosis is confirmed.

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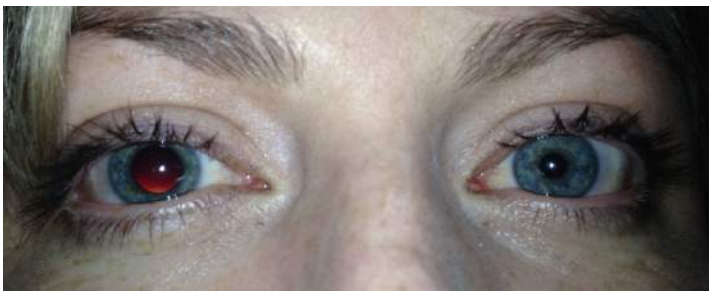

Figure. Photograph of 33-year-old female presenting with asymmetric pupils.

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Visual Hallucinations: Charles Bonnet Syndrome

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The following is a case of Charles Bonnet syndrome in an 86-year-old woman who presented with visual hallucinations. The differential diagnosis of visual hallucinations is broad and emergency physicians should be knowledgeable of the possible etiologies. [West J Emerg Med. 2012;13(6):544-547]

INTRODUCTION

Visual hallucinations are not a common presentation in the emergency department (ED). However, emergency physicians should be adept in differentiating between various causes. Patients who present with this neuro-ophthalmologic dysfunction carry a broad differential diagnosis. This usually results in an extensive evaluation, including neuro-imaging, laboratory testing, and consultation with pertinent specialties, such as neurology or psychiatry. While there are many serious causes of visual hallucinations, in this case report we present an unusual yet benign etiology for visual hallucinations.

CASE REPORT

An 86-year-old woman presented to the ED with a chief complaint of visual hallucinations. She had been having these "visions" for a week, occurring mainly in the evening. The hallucinations were episodic, binocular and lasted for hours. She was unable to identify any specific factors that would trigger their arrival or resolution.

The hallucinations were clear and vivid, representing either various objects around the house moving, such as a clock and a planter, or people and faces on the ceiling or corner of her room. She had become so frightened thinking she was "going crazy" that she decided to come to the ED. While experiencing these hallucinations, the patient had no change in mental status and maintained full insight and awareness. She denied any other visual disturbances, such as diplopia, scotomata, tunnel vision or loss of vision. She further denied auditory or other sensory hallucinations as well as headaches, fever, or trauma. There was no history of drug or alcohol abuse.

Her past medical history included open-angle glaucoma, macular degeneration, cataracts, hypothyroidism, hyperlipidemia and gastric reflux. Her surgical history was relevant for bilateral cataract surgeries with her right eye requiring 2 surgeries, as well as a corneal transplant. She denied any psychiatric history.

On physical examination she appeared well and in no acute distress. She was oriented to person, place and time. Her vital signs were: blood pressure 213/98 mmHg, pulse rate 66 beats/minute and regular, respirations 20 breaths/minute, temperature of 36.8°C and oxygen saturation of 97% on room air. There were no complaints of any hallucinations at the time of the exam. She had bilateral iridectomies and her corrected visual acuities were 20/50 OD and 20/100 OS. The visual fields were full, and her fundoscopic exam revealed no gross abnormalities. Neurologic examination showed no focal findings. A mental status examination did not reveal mood disturbance or cognitive deficits, and she scored 30/30 points on her Mini Mental Status Exam (MMSE). The remainder of her physical examination was normal.

Laboratory studies demonstrated a serum sodium level of 128 mEq/L and a serum chloride of 92 mEq/L. Glucose and renal function were normal. White blood cell count was 8.3 x 10^9/L. A urinalysis was positive for nitrites, 2+ leukocyte esterase and 50-100 white blood cells/HPF. Computed tomography (CT) of the head without contrast showed no acute intracranial hemorrhage, stable moderate to marked chronic microvascular ischemic changes in the supratentorial white matter, and a stable small chronic infarct in the right basal ganglia.

Based on clinical history and exam, a diagnosis of Charles Bonnet syndrome was made by the ED attending physician. She was seen 2 days later by her ophthalmologist who concurred with the ED diagnosis based on similar presentation of many previous patients in his practice. At the time of her follow up, the hallucinations had resolved and her eye exam was unremarkable.

DISCUSSION

There are many etiologies for visual hallucinations, which include retinal disease, migraines, acute stroke, drug-related side effects, neurodegenerative disease, alcohol and/or drug use, toxic-metabolic encephalopathy and psychiatric illness. Visual hallucinations can be categorized as simple or complex. Simple hallucinations involve basic imagery, such as lights, colors, lines and shapes, whereas complex hallucinations include images of people, objects or specific scenes. Classifying the type of visual hallucinations can help narrow the differential diagnosis, thereby aiding in accurately diagnosing the patient's underlying condition. Other features that should be delineated are the presence versus absence of insight, monocular versus binocular involvement, amount of visual field involvement, triggers, frequency and duration.

Charles Bonnet syndrome is an uncommon condition causing visual hallucinations in patients without mental illness. Charles Bonnet, a Swiss philosopher, first described the syndrome in 1760 in a publication describing visual hallucinations experienced by his grandfather who was blind secondary to cataracts.[1] However, it was not until 1967 that a Swiss scientist, George de Morsier, labeled the condition as Charles Bonnet syndrome.[2] Since then, a number of case reports have been described in the ophthalmology and psychiatry literature.[3]

The neurophysiology explaining the visual hallucinations in Charles Bonnet syndrome is not clearly elucidated. The currently accepted theory suggests that vision loss leads to visual sensory de-afferentation, causing disinhibition, and later spontaneous firing, of the visual cortical regions.[7] Visual deprivation experiments have resulted in similar hallucinations, and functional magnetic resonance imaging (MRI) in patients with Charles Bonnet syndrome has found an association with visual hallucinations and spontaneous activity of the ventral occipital lobe.[8]

The majority of patients with Charles Bonnet syndrome are elderly with a mean age of 70–85 years. However, cases have been reported in all age groups. Identifying the exact prevalence of Charles Bonnet syndrome is difficult, and the number differs widely among various studies.[3,4] This uncertainty and inconsistency is likely due to two main causes: underreporting of symptoms and misdiagnosis. Up to 60% of patients with Charles Bonnet syndrome are hesitant to tell their physician about their visual hallucinations for fear of being labeled with a mental illness or dementia.[6] Misdiagnosis is also common as the syndrome is not recognized by clinicians and often labeled as psychosis, delirium or early dementia.

Patients with Charles Bonnet syndrome are also likely to have a history of diminished visual acuity or visual field loss. Both this and elderly age were found to be the primary factors correlated with Charles Bonnet syndrome.[5] Visual deficits are generally the result of macular degeneration, cataracts, glaucoma or diabetic retinopathy. Patients possess insight into the unreality of their visual experiences, which are commonly pleasant but may cause significant anxiety.

The visual hallucinations of Charles Bonnet syndrome may be simple or complex. The characteristic features of these images are not associated with the specific anatomical location of the ocular injury. However, they will typically correspond to the area of visual loss, whether that localizes to a certain visual field or are monocular versus binocular.[9] These images are typically well-defined, clear, and lack personal meaning or impact to the patient. Hallucinations will occur more frequently with eyes open and will disappear when the eyes close or visual gaze changes.[3] One study also showed that hallucinations appeared more often during periods of sensory deprivation, such as inactivity or evening and nighttime.[9] The duration of hallucinations will vary from continuous to less than one minute, but most patients report duration of several minutes occurring multiple times a day or week.[7]

Symptoms caused by Charles Bonnet syndrome cannot be explained by the presence of a psychiatric disorder, and patients do not have any significant metabolic derangements or impaired sensorium that would otherwise explain the symptoms. Although Charles Bonnet syndrome has no official diagnostic criteria, the features described are generally accepted and can be used as guidelines to make the diagnosis when appropriate (Table 1). A patient presenting to the ED with symptoms suggestive of Charles Bonnet syndrome should receive basic laboratory tests, such as a metabolic panel and complete blood count along with neuro-imaging, typically with CT without contrast. MRI could be considered, although it would likely be most appropriate in the outpatient setting.

Our patient had the characteristic features with her age and history of vision loss. She also had full insight into the fictional nature of her hallucinations and met the diagnostic criteria of Charles Bonnet syndrome discussed previously. However, our patient also presented with hyponatremia and a urinary tract infection, although these findings were unlikely to be the cause of her symptoms. At a level of 128 mEq/L, her hyponatremia would typically be considered mild. Most neurological manifestations of hyponatremia occur at levels of < 115 mEq/L, and it would be unlikely for a patient with a serum sodium of 128 mEq/L to exhibit visual hallucinations

Table 1. Diagnostic criteria for Charles Bonnet syndrome.[3]

Diagnostic criteria
At least one complex visual hallucination within the past 4 weeks
A period between the first and the last hallucination exceeding 4 weeks
Full or partial retention of insight into the unreal nature of the hallucinations
Absence of hallucinations in other sensory modalities
Absence of delusions

without other changes in mental status as the only symptom of her hyponatremia. The urinary tract infection was a subclinical diagnosis as our patient had no symptoms. She also had no risk factors of a complicated infection, such as a recent catheterization, residence in a nursing home, or an immunocompromised status. In contrast, she was a high functioning and independent octogenarian. In these circumstances, it would be unlikely that the infection was the primary cause of the patient's visual hallucinations with no other neurologic changes or confusion. However, it is possible that these abnormalities could have contributed in exacerbating her symptoms, although this would be difficult to fully assess.

Currently, there is no universally accepted treatment for Charles Bonnet syndrome. Visual hallucinations often resolve once the underlying cause of vision loss is rectified. Unfortunately, in some cases, they can be persistent for several years.[10] In these scenarios, patients can learn how to suppress hallucinations and improve quality of life. Clinicians can encourage patients to heighten visual stimulation, either with increased arousal through social interaction or activity or rapid eye movements, both of which can reduce hallucinations.[2,11]

Anecdotally, typical and atypical antipsychotics have shown benefit in individual patients, although there is little evidence to support this and may not outweigh the risks of side effects or interactions with other medications.[4] Atypical antipychotic medications such as Risperidone (Risperdal), Quetiapine (Seroquel) and Olanzapine (Zyprexa) have been used with varying success.

Often times, the cause of the hallucinations is more distressing to the patient than the actual hallucinations, and therefore reassurance from the provider can make patients more comfortable without pharmacologic treatment.[13]

Once the diagnosis of Charles Bonnet syndrome is made in the ED, the provider can reassure the patient and provide suggestions to suppress hallucinations as mentioned previously. Any metabolic abnormalities should be corrected to prevent potential exacerbations of symptoms. Referral to an ophthalmologist for follow up would be appropriate, especially if visual acuity correction is needed. Patients can typically be discharged safely without any prescriptions and follow up with their primary care provider to assess whether further diagnostic work up, such as MRI or a trial of an antipsychotic medication, would be necessary.

Another closely related diagnosis that should be considered in patients that present with acute onset of visual hallucinations is Lhermitte's hallucinosis, otherwise known as peduncular hallucinosis. Similar to Charles Bonnet syndrome, Lhermitte's hallucinosis is more commonly attributed to the elderly. The visual hallucinations in Lhermitte's hallucinosis are vivid, well-formed, and usually are recognized to be unreal. In contrast though, Lhermitte's hallucinosis is associated with dementia, with correlated lesions or infarcts in the midbrain or brainstem on neuro-imaging.[13] Also, many patients will demonstrate other neurological findings, reflecting deficits from prior cerebrovascular injuries. In a case series of 5 patients with Lhermitte's hallucinosis, all patients were found to have decreased MMSE scores, and patients had intermittently displayed periods of confusion and disorientation. Furthermore, visual hallucinations were accompanied by various deficits in higher cortical functioning, including memory deficits, agnosia and dysarthria.[13,14] Although our patient did not undergo a MRI, the absence of any cognitive deficits or episodes of confusion made this diagnosis unlikely. Lhermitte's hallucinosis should be included in the differential when evaluating patients similar to the one described in this case report.

CONCLUSION

Charles Bonnet syndrome should be considered in all elderly patients who present with visual hallucinations. However, more serious causes of hallucinations should be considered and ruled out, as Charles Bonnet syndrome is a clinical diagnosis of exclusion. As our society ages, EPs are likely to encounter this diagnosis with greater frequency as vision problems such as cataracts and macular degeneration become more common. Although Charles Bonnet syndrome is benign, the visual hallucinations can cause great anxiety for patients. Therefore, increased awareness of this condition can assist clinicians in proper diagnosis, counseling and reassurance of their patients.

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Increase in Non-Contrast Computerized Tomography Scans of the Head Following Popular Media Stories About Head Injury

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Introduction: On March 18, 2009, actress Natasha Richardson died after a head injury. It is possible that the rate of patients presenting with mild head injury and receiving computed tomographies (CTs) may have been influenced by the Richardson event. We hypothesized that there was a statistically significant increase in the rate of census-adjusted head CTs performed for mild trauma after March 16, 2009, compared to prior to this date.

Methods: We included all with a non-contrast head CT performed from the emergency department (ED) between March 1and April 15, 2009, for minor trauma. The primary outcome was the census-adjusted rate of head CTs per time (# of head CTs/census). We compared the census adjusted rate for the 2 weeks prior to 2 weeks after the accident. To document media dissemination we searched Lexis-Nexis for news stories mentioning "Richardson."

Results: In the 2 weeks prior to March 16, 2009, the census-adjusted rate was 0.81% (95% CI 0.54–1.16) and there were no stories. The first media reports appeared on March 16, 2009, (n = 19) and quickly doubled (n = 40, n = 43) over the subsequent 2 days. The rate of CTs nearly doubled during the 2 weeks post accident 1.46% (1.10–1.91%). This absolute increase in rate percentage was statistically significant. (0.65%; 0.17 to 1.14%).

Conclusion: The percentage of all ED patients seen with mild trauma tested with head CT almost doubled when comparing the pre-Richardson accident vs. post time periods. There was an increase in media reports of the accident that occurred rapidly after the event and peaked on day 3. [West J Emerg Med. 2012;13(6):548-550]

INTRODUCTION

The number of visits and length of stay in emergency departments (ED) are increasing, and so are computerized tomography (CT) use.[1-4] CT of the brain is an important tool for diagnosing significant traumatic brain injury, but overuse is concerning due to the resultant increase in healthcare costs and risks of exposure to ionizing radiation.[5]

At the same time, the public increasingly uses popular media as a source of healthcare information. This has been shown to be true both for people actively seeking information about a particular topic and for those who passively receive information relating to health.[6] Although media may be used as a source of healthcare information, the ability of the national media to report on health topics in proportion to their risk to public health has been called into question in prior studies.[7]

Stories in the national media relating the health problems of celebrities are common in both print and broadcast media. Celebrity involvement in a particular malady has been shown to increase media attention to that topic, as has been particularly well demonstrated with cancer.[8] Anecdotally, healthcare providers at our institution reported an increase in the number of patients requesting CT of the head after minor head injury in the days and weeks following the story of

actress Natasha Richardson's tragic death on March 18, 2009. Ms. Richardson suffered what may have been an epidural hematoma after a blow to the head sustained while skiing. The story received wide attention in the national media with some outlets characterizing her condition as the "talk and die" or the "walk and die" syndrome.[9-10]

It may be hypothesized that some patients who sought care for mild head injury in the days and weeks following Ms. Richardson's injury were influenced by media sources. Although there is some evidence to suggest that celebrity health issues have an impact on the health-associated behaviors of the general public,[11] it is yet unclear to what effect media reports on healthcare-related topics have on short-term emergency healthcare use by the public.

We hypothesized that there would be an increase in the census-adjusted rate of head CTs (CARHCT) performed at our institution and that this increase would show some relationship to media coverage of Ms. Richardson's injury.

METHODS

This was a retrospective chart review approved by the Institutional Review Board at Northwestern. We searched the electronic medical record database (Enterprise Data Warehouse, or EDW) at Northwestern Memorial Hospital to find patients who had a head CT performed from the ED between March 1, 2009 and April 15, 2009. We then performed chart review to exclude any patient meeting exclusion criteria in Table. Patients not excluded were considered to be those for whom a head CT had been ordered from the ED for minor head trauma and were included in the final analysis. A total of 662 patient encounters met initial screening criteria, and of those 123 were included in the final analysis. The most common reasons for exclusion were atraumatic headache and mention of a focal neurological deficit. We also collected data on daily ED census during the time period in question.

We performed statistical analysis to determine the CARHCT per day. The month following Ms. Richardson's injury was divided into 15-day sections, and we compared the rate of CARHCTs to the 16 days prior to the injury.

To determine the frequency of media coverage of Ms. Richardson's accident and its effect, we extracted a raw count of stories about Natasha Richardson from a purposive sample of national newspapers, network television stations, a cable news network, and a radio network. Frequency in this research was a corollary for amount of media coverage. The purposive sample was designed to represent those outlets with the largest audiences. They were the four major United States newspapers (*New York Times*, *USA Today*, *Wall Street Journal*, and *Washington Post*), the morning and evening news programs of the four national networks (ABC, CBS, NBC, and FOX), one cable news network (CNN), and one national radio network (NPR).

The unit of analysis was the individual story, defined as a news or opinion piece and excluding paid death notices,

Table. Exclusion criteria of patients in the study.

Exclusion criteria
Age less than 18 years
Age greater than 60 years
Temperature greater than 38.1C
Systolic blood pressure less than 90
Heart rate less than 50
Heart rate greater than 100
Respiratory rate less than 8
Oxygen saturation less than 92%
Mention of any intoxication
Trauma team activation
Stroke team activation
Atraumatic headache
Focal neurologic deficit
Anticoagulant medication (aspirin, clopidogrel, heparin, low-molecular weight heparin, warfarin)
Known prior intracranial pathology

in the Lexis-Nexis Academic Universe database mentioning "Richardson" anywhere in the text or transcript. Stories that were not about Natasha Richardson (Bill Richardson, governor of New Mexico, for example) were then excluded from the data set, which ran from March 1, 2009, to April 15, 2009 (n = 193). Data collected before the accident established a baseline of media coverage.

RESULTS

Figure 1 shows the CARHCT and the number of media stories. CARHCT in the 2 weeks leading up to the accident was 0.81% (95% confidence interval [CI] 0.54–1.16). In the 15-day period following the accident CARHCT was nearly double at 1.46% (95% CI 1.10–1.91). In the next 15-day period the CARHCT was 1.13% (95% CI 0.80–1.55). Absolute increase in CARHCT for the 30-day period prior to the accident compared to the period after was statistically significant (0.65%; 95% CI 0.17 to 1.14%).

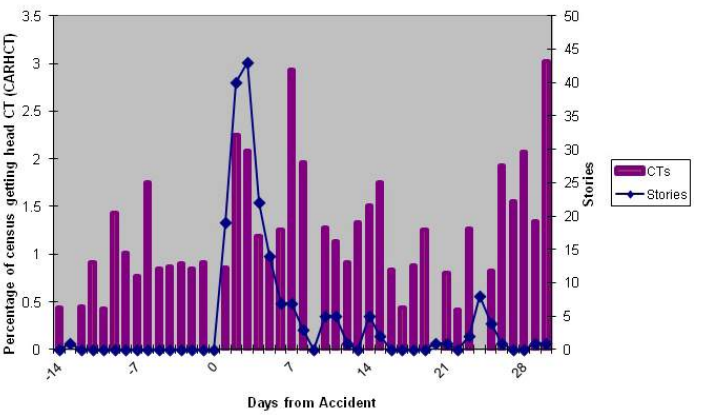

Figure. Percentage of census getting head computed tomography.

Media reports about Ms. Richardson's injury peaked on March 19, 2009, and quickly decreased over the next few days. During that time there was still an increase in CARHCT. All 10 news outlets covered the Richardson story, although frequency of coverage varied significantly. Only 1 story appeared in the *Wall Street Journal* to a high of 74 stories on CNN. Coverage peaked on March 19, 2009, at 43 stories.

DISCUSSION

We demonstrated a statistical increase in the CARHCT in a single ED in the immediate period following heavy media coverage of a celebrity injury. While similar work has been done documenting this increase in both CT and ED use, this is the first study to show a temporal relationship between media coverage and ED use.

It is unclear to what extent this increase in CARHCT was due to patient factors, clinician factors or both. Few data exist as to what extent such media attention has on clinician behavior, although it may function as a kind of bias.

Our data suggest that patient and clinician behavior continues to be affected by media stories even as the story becomes less active. More work would need to be done to explore this relationship.

LIMITATIONS

Our study has several limitations. It is retrospective and single centered. In addition, while we designed exclusion criteria meant to exclude CTs performed for obvious reasons other than minor head trauma, we did not analyze the individual clinical decisions leading up the ordering of a brain CT. It is possible that for some of the encounters in our sample neither the clinician nor the patient had ever heard of Ms. Richardson, and her accident had no bearing on their interaction. On the media side of the project, a simple count of stories about Ms. Richardson has its own set of limitations. We did not do story-level analysis for content, and as such some stories may have made little or no mention of her accident.

CONCLUSION

There was a statistically significant increase in head CT utilization in our ED following media coverage of a celebrity head injury.

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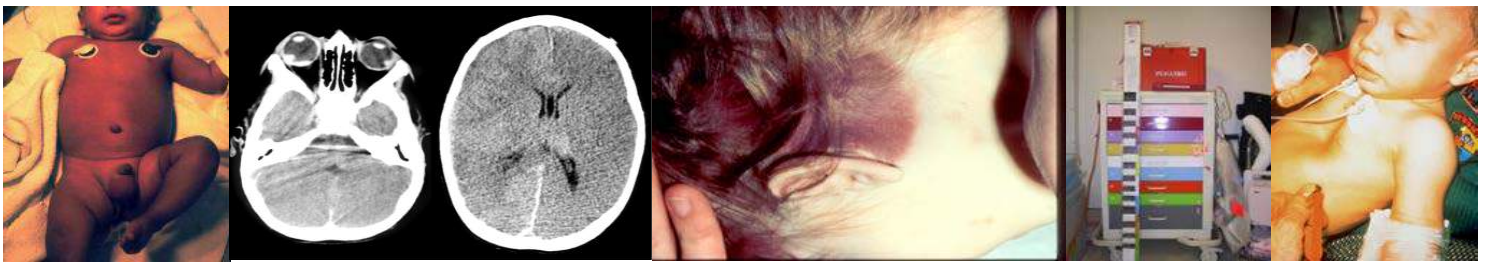

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